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CHRONIC SINUSITIS

THE ENDEMIC FOCUS AND CARRIER OF THE COMMON COLD

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SAN FRANCISCO

It is necessary to trace the development of the morbid changes associated with chronic sinusitis and to follow the clinical behavior of the disease to appreciate its association with the origin and epidemicity of the common cold.

CHRONIC NONINFECTIOUS INFLAMMATION

Allergy and biochemical factors may build in the sinus an edematous, reticulous type of mucosa favorable to subsequent bacterial invasion and their protracted establishment.[1]

ACUTE INFECTIOUS INFLAMMATION

The True Cold.—Etiology: It is granted that the so-called complications of the common cold are caused by the essential pathogens.[2] The fact that a cold can be caused by a filtrable virus does not deny the same etiologic power to the hemolytic streptococcus.

Morbid Anatomy: The changes that take place in the tissues depend on the character of the mucosa at the time of the infection. A normal mucosa independent of allergic or biochemical disturbances reacts dif-

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1. Sewall, E. C.: Sinusitis Allergy and the Common Cold: Conception of Their Relationships, Arch. Otolaryng. **22**:425 (Oct.) 1935. Coates, G. M., and Ersner, M. S.: Occurrence of Eosinophils in the Mucous Membrane of the Maxillary Sinus in Asthmatic Patients, ibid. **11**:158 (Feb.) 1930. Hansel, F. K.: Clinical and Histopathologic Studies of the Nose and Sinuses in Allergy, J. Allergy **1**:43 (Nov.) 1929.

2. (*a*) Zinsser, Hans, and Bayne-Jones, Stanhope: A Textbook of Bacteriology with a Section on Pathogenic Protozoa, New York, D. Appleton-Century Company, Inc., 1934. (*b*) Webster, L. T., and Clow, A. D.: Association of Pneumococci, Hemophilis Influenzae, and Streptococcus Hemolyticus with Coryza, Pharyngitis, and Sinusitis in Man, J. Exper. Med. **55**:445 (March) 1932. (*c*) Kneeland, Y., Jr., and Dawes, C. F.: Studies on the Common Cold: Relationship of Pathogenic Bacteria to Upper Respiratory Disease in Infants, ibid. **55**:735 (May) 1932.

ferently to infection than a mucosa that has been altered by these factors and may still be under their malign influence.

1. The normal mucosa of a sinus is very thin.[3] Its structure offers comparatively little food for bacterial growth. Its inflammation, therefore, is mild and of short duration, and it rarely becomes chronic. This is spoken of as a light cold. Increased fibrosis is the result of inflammation.[4] The mucosa is left more densely fibrous by each succeeding attack. A person with a normal mucosa has fewer and milder "colds" as age advances. When, by an unusual combination of circumstances, this type of mucosa becomes chronically diseased, purulent sinusitis results.

2. The altered sinus mucosa is thickened and essentially edematous. The fibrous structure is reticulous. Bacteria find it a favorable medium. Their growth may be favored by allergic or biochemical factors. The inflammation is protracted and severe—a "bad cold." Each attack increases the reticular fibrosis. Chronicity tends to supervene. Chronic hyperplastic sinusitis results.

CHRONIC INFECTIOUS INFLAMMATION

Chronic Purulent Sinusitis.—Etiology: Chronic purulent sinusitis is the result of acute infectious inflammation of a normal mucosa. Unusual factors must conspire to overcome the inherent resistance of this tissue.

Morbid Anatomy: The thinness of the mucosa is the determining factor in the resulting tissue changes. There is no protective edema, and the bone and the epithelium feel the brunt of the bacterial activity which traumatizes them. The tender, ciliated epithelium is changed into the protective pavement type. Its surface becomes smooth and glazed. The nonmucous, purulent discharge dries quickly and forms crusts, which in turn traumatize the mucosa. Atrophy is characteristic of this type of sinusitis. It affects not only the mucosa but the underlying bone. Atrophic rhinitis is caused by chronic purulent sinusitis.

Chronic Hyperplastic Sinusitis.—Etiology: This is the common type of sinusitis, the source and carrier of the "common cold." It is the result of acute infection of an altered mucosa.

Morbid Anatomy: The bacteria gain entrance to the submucosa during an acute common cold. They find conditions favorable, and after a protracted, stormy acute stage, they are forced into a semiquiescent state by the body defense. They remain in the depths of the tissues. Their virulence has been reduced, as shown by their indolent behavior. They resemble the noninfectious irritants in their effect on

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3. Seminov, Herman: The Surgical Pathology of Sinusitis, Tr. Pacific Coast Oto-Ophth. Soc. **22**:125, 1934.

4. Willmaack, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1936.

the tissues. They lose their chemotactic activity. The polymorphonuclear leukocyte is replaced by the mononuclear or the eosinophil. The discharge may be reduced to a minimum and be clear to the naked eye. Microscopic examination, however, will always disclose cytologic and perhaps bacteriologic evidence of disease. The chronic sinusitis is now in the quiescent, more or less protracted latent state characteristic of this disease.

The False Cold.—The latent period of chronic sinusitis is varied by certain phenomena which give rise to symptoms resembling a "true cold." Such an attack, which usually can be differentiated clinically, may be called a "false cold." [5] Chilling of the body is the outstanding physical factor responsible for the attack. Primarily it is a vasomotor disturbance. Chilling causes a spasm of the vessels [6] of the submucosa which, with the subsequent relaxation, favors stasis and encourages activity in the bacteria lurking in the tissues. Bacterial quickening leads to an immediate increase in the nasal discharge. The resumption of chemotactic activity colors it by an accumulation of leukocytes. There is increased toxic absorption because of bacterial activity and the pressure generated by the swelling of the hyperplastic mucosa and accumulation of discharge in the sinus cavity. Fulness in the head may increase to an intolerable headache or neuralgia. These symptoms constitute the false cold, which is commonly mistaken for the true common cold. It is differentiated by the fact that it confers no immunity to such attacks. All the symptoms are milder than those to which the patient is accustomed in his yearly or semiyearly true cold. It is also characterized by being brought on immediately and repeatedly by chilling and exposure. It is further distinguished by rapidly clearing up on return to mild atmosphere and body warmth. The sudden disappearance of this type of cold often occasions surprise. Not infrequently a false value is attributed to some remedy that happened to be employed.

Such a vasomotor exacerbation may occur with greater or less frequency in persons suffering from chronic hyperplastic sinusitis. The frequency and severity of the attacks are directly proportional to the inclemency of climatic conditions.

There is a tendency to discourage the use of the word "cold" because of the doubtful relationship of cold to infection. The word appears in many languages and signifies the accumulated experience of the centuries: German, *Erkältung;* French, *refroidissement;* Italian, *rafredorre,* and Spanish, *resfrio.* The subject of cold cannot be lightly dismissed without granting the relationship of chilling to the fre-

5. Sewall, E. C.: External Operation on the Ethmosphenoid-Frontal Group of Sinuses Under Local Anesthesia, Arch. Otolaryng. **4**:377 (Nov.) 1926.

6. Schmidt, P.: Erkältung und Tuberkulose, Tuberkulose **15**:197 (July 10) 1935.

quent vasomotor exacerbations of chronic sinusitis which in the past have not been distinguished from the "common cold." Such fake colds have so dominated the picture historically that the word cold has come to designate nasal conditions of varied etiology. It is an answer in contradiction to those who believe in the modernity of sinusitis. The person with chronic sinusitis learns to fear drafts, cold and exposure. The normal person respects them only from hearsay. He has no personal experience with their evil effects. Such a person is indifferent to chilling and exposure. He catches cold only when it is brought to him. He may then experience chilly sensations, often independent of environment.

The exacerbations are more frequent and more marked in youth. Children are often said, erroneously, to have just one cold after another or to have a continuous cold.

The mucosa gradually becomes more hyperplastic with the years. The resistance of the body to the chemotactic activity of the bacteria increases. In consequence, those who have had this form of sinusitis for many years usually have comparatively few leukocytes in the discharge. They often have nothing but a meager, occasional dropping of clear water from the nose. The true condition is often missed on that account. According to Nelson and his co-workers:[7] "In addition to pollen concentration, weather changes are important in precipitating attacks of asthma in pollen sensitive patients. Cooling or filtered air may aggravate asthma because of increased relative humidity." Chilling is probably the cause of the symptoms described, and the asthma in such cases is doubtless associated with chronic hyperplastic sinusitis.

IMMUNITY

After an acute cold the chimpanzee is refractory for about four months[8] and man, for six months or a year or longer. This immunity is just as definite as that from scarlet fever. There are exceptions in both instances. One or two colds a year is the determined average.

Such definite immunity to the acute common cold is granted equally to a person with chronic sinusitis. If this were not true there would be no chronic sinusitis. In its development, a definite immunity checks the acute process and allows the bacteria to be tolerated in the tissues, where they may remain permanently.

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7. Nelson, T.; Rappaport, B. Z., and Welker, W. H.: Effect of Air Filtration in Hay Fever and Pollen Asthma: Further Studies, J. A. M. A. **100**:1385 (May 6) 1933.

8. Dochez, A. R.; Mills, Katherine C., and Kneeland, Yale: Diseases of the Upper Respiratory Tract: Problems Connected with Etiology and Prophylaxis, J. A. M. A. **101**:1441 (Nov. 4) 1933. Dochez, A. R.: Limited Consideration of Certain Aspects of Acute Infection of the Respiratory Tract, Medicine **12**:245 (Sept.) 1933.

Vasomotor exacerbations or false colds may occur in chronic sinusitis, varying in frequency and severity with the chilling, biochemical influences and the extent of the morbid changes. A true cold will not develop until immunity disappears; when that occurs the bacteria in the tissues are no longer controllable when stimulated by the vasomotor disturbances described. As soon as the defense is weakened, the bacteria gain virulence with their increased activity. They are not saprophytic or commensal germs. They occupy the tissues and are in a position to develop as they might in the body of a laboratory animal. They get out of hand, and then no amount of body warmth will control them. A true cold thus develops in a person with chronic sinusitis who may have had no outside contacts whatever.

THE ENDEMIC FOCUS OF THE COMMON COLD

From this source or carrier a local or a general epidemic may arise. Without the ever replenished reservoir afforded by chronic sinusitis, there would be no true common colds.

During the summer there are comparatively few colds. This conforms to the fact that the person with chronic sinusitis is comparatively free from exacerbations during the warm weather. With the first drop in the temperature there is often a greatly increased incidence, and this is augmented by the progress of winter. It often gains epidemic proportions. This is recognized by primitive people, and some have a word to describe harmful or helpful weather. The "Kona" of the Hawaiian Islands is an example of the one, while the "Chinook" of the Oregon country is an example of the other.

Chronic sinusitis was essentially a disease of community life and the temperate zone. Communication has changed conditions so that there are few isolated regions where primitive conditions remain. Once the tropics were comparatively free. Rheumatic fever and its sequelae and the arthritides were then significantly less prevalent. Before the approach of civilization the arctic zone was endemically free from cold. The extreme low temperature discouraged the perpetuation and spread of infection.[10] The lack of immunity, however, made the Eskimos an extremely susceptible people when they came into actual contact with the common cold. In from twenty-four to seventy-two hours after the arrival of the first ship in the spring, the arctic community regularly succumbed to an epidemic of common cold. This occurred even when there was no person with an acute cold on the ship. The explanation obviously would appear to be that while there were no persons with

9. Footnote deleted by the author.

10. Paul, J. H., and Freeze, H. L.: Epidemiological and Bacteriological Study of "Common Cold" in Isolated Arctic Community, Am. J. Hyg. **17**:517 (May) 1933.

acute cold, there were persons with chronic sinusitis, the carriers of the common cold. Persons on ships at sea, away from port and free from colds for months, and isolated groups have been known to have epidemics of colds.

A significant occurrence is reported of a company of arctic explorers who, after having been away from civilization and free from colds for many months, encountered an Eskimo tribe. The natives at once contracted colds of epidemic distribution. The explorers also then experienced a similar outbreak of colds among themselves. The explanation of this phenomenon would appear to depend on the pandemic character of chronic sinusitis among civilized people and its ability to transmit the common cold. Statistical incidence would indicate that a certain number of the explorers had chronic sinusitis. The extreme low temperature discouraged an outbreak of "cold" among those accustomed to civilization's flora. The Eskimos lacked all immunity and fell quick victims to the infection. In such fertile soil the bacteria or other causative agent acquired sufficient virulence to overcome the resistance of the carriers themselves. They were than "hoist on their own petard."

STREPTOCOCCUS: ITS ENDEMIC FOCUS

In the foregoing paragraphs, for the sake of accuracy, the terms bacteria and virus have described the etiologic factors in the common cold.

A filtrable virus appears to be established as the cause of the common cold. A virus was erroneously believed to be the cause of measles. It is difficult to ignore the hemolytic streptococcus in considering the etiology of the common cold. The importance of the virus does not deny an equal importance to the streptococcus itself. Swine influenza is transferable only by a virus plus bacteria. Perhaps the same is true in a common cold, as the streptococcus is practically ubiquitous in crowded community life. This may explain the impotence of the virus when sent to England. At any rate, the infective substances, whatever they may be, that cause the dissemination of a common cold arise in association with the activity of the streptococcus in an acute exacerbation of chronic sinusitis.

The hemolytic streptococcus enters the nose and invades the mucosa of the accessory sinuses. The mucosa is continuous, and the simultaneous and contiguous involvement is much too intimate to be called a complication. Still it is universally acknowledged that bacteria are the cause of these so-called complications.

The reaction of the tissues produces a discharge. The results of bacterial examination of this discharge may be misleading. The streptococcus may not leave the tissues in great numbers, and even if they do and accumulate in the sinus cavity, they are crowded out by more

readily growing bacteria. This lack of competitive power of growth is shown in laboratory culture. It is not true in the tissues. There the hemolytic streptococcus reigns supreme.

The hemolytic streptococci located in the tissues produce the active and complicated process known as the common cold. They are usually controlled in a comparatively short time by the development of an immunity which allows the tissues to tolerate their presence if the acute cold becomes chronic sinusitis. As the acute process fades, the hemolytic streptococci disappear and are replaced in the tissues by the nonhemolytic type of organism. Streptococcus viridans is usually found in chronic processes and apparently represents the dormant stage of the streptococcus. This is true not only in the sinus but in the tissues of the tonsil, heart and elsewhere. Diseases that are obviously caused by the hemolytic streptococcus continue in the chronic state under the insidious influence of Str. viridans.

Str. viridans is furnished regularly to the throat through the ever-present discharge from the diseased sinus. Again, however, the organisms of this species may be crowded out by the other, more easily growing bacteria and not be found on culture from the throat. They may, as may the hemolytic streptococci and other bacteria, reach the throat from outside sources. The germs are spread about very freely by the multitude of persons with sinusitis. This is exemplified by the baby's throat, which is sterile at birth and gradually acquires the common flora in a few weeks without having an acute infection.

All of the many excellent studies of the flora of the throat have been unable to account for the variation in the presence of streptococci and other bacteria because they have failed to distinguish between health and disease. Those with chronic sinusitis are chronically infected. They are of common incidence and must be considered separately in any bacteriologic or other statistical study.

Str. viridans is not a vigorously growing germ. It dies in saliva in a few weeks.[2a] The accidental introduction from without does not satisfactorily account for its commensal and often almost universal presence in the saliva of the throat. It comes chiefly from the adjacent diseased sinus. Diseased teeth and tonsils may furnish their quota. However, these are usually secondarily infected from the sinus. The tonsils are covered even down to the bottom of the crypts with squamous epithelium. The bacteria penetrate this armor only under the influence of ulcerative processes so plainly seen in the morbid structures of a chronically diseased tonsil. The delicate susceptibility of the mucosa of the sinus contrasts convincingly. Probably neither Str. haemolyticus nor Str. viridans lying in a commensal state in the saliva causes disease except in fanciful association with the filtrable virus as suggested. The

protective epithelium of the mouth and throat is rarely penetrated, and even an abrasion of the surface rarely becomes the seat of a streptococcic infection.

Acute disease arises from the streptococci lurking in the mucosa of the sinus. They are stimulated to activity by biochemical irritation and by chilling. These cause vasomotor exacerbations of chronic sinusitis or false colds. There may be an increase of Str. viridans in the nose and throat.

If conditions are favorable and the immunity of the host has expired, the bacteria may get out of hand. Str. viridans reverts to the virulent hemolytic type, and the true common cold occurs. The common cold may thus develop when immunity has run out in a person with chronic sinusitis without any outside contacts whatever. It is capable of spreading the infection to others. The hemolytic streptococci may or may not be increased in the nose and throat, depending on the activity of the more easily growing bacteria.

The change of Str. viridans into the hemolytic type and vice versa, so obvious clinically, is difficult to demonstrate experimentally. However, it has been done repeatedly, even in culture. Its demonstration in animal tissues has been criticized as susceptible to error. Such conditions are more comparable to clinical experience than laboratory culture.

Gay,[11] in his recent textbook, said:

> The possible interchange, transformation or mutation of the relatively constant hemolytic strains of streptococci to viridans, and a similar conversion of streptococci to pneumococci and the reverse processes must now be seriously considered if not in part accepted.

The character of the streptococcus supports the theory of transmutation. Although a number of specific diseases are attributed to it, none of them are associated with a strictly invariable strain. The fibrinolytic activity varies with the virulence of the strain.[12] The streptococcus is intimately associated with man, and its source is the chronically infected sinus. Birds are immune to it and most other animals are less susceptible. The surgeon has long known the comparative freedom from postoperative infection in isolated districts. The airplane has permitted a study that has shown that the streptococcus disappears comparatively quickly as the altitude is increased.

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11. Gay, F. P., and others: Agents of Disease and Host Resistance Including the Principles of Immunology, Bacteriology, Mycology, Protozoology, Parasitology and Virus Disease, Springfield, Ill., Charles C. Thomas, Publisher, 1935.

12. Madison, R. R.: Fibrinolytic Staphylococci, Proc. Soc. Exper. Biol. & Med. **33**:209 (Nov.) 1935.

The importance of chronic sinusitis in the spread of puerperal fever has been recognized, but practically nothing has been done to exclude the streptococcus carrier. He can be easily recognized from the fact that chronic sinusitis furnishes discharge, and a normal person has no such discharge. This can be substantiated cytologically. Bacterial contaminants are likely to mask temporarily the presence of streptococcus.

SUMMARY

1. Noninfectious factors favor chronic sinus infection.

2. The common cold depends for its severity or benignity on the character of the mucosa.

3. The etiologic significance of Str. haemolyticus is not denied by the importance of a virus.

4. The "false cold" is an exacerbation of chronic sinusitis. It confers no immunity.

5. The "true cold" may occur in a person with chronic sinusitis without any outside contacts. He may thus start an epidemic.

6. The true cold confers a definite immunity of from six months to a year.

7. The chronically infected sinus is the endemic focus and carrier of the common cold.

8. The chronically infected sinus is the source of the streptococcus.

ABSTRACT OF DISCUSSION

DR. VIRGIL W. MCCARTY, Kansas City, Mo.: Dr. Sewall's contribution to the literature on the pathologic conditions of the nose is too well known and too valuable for me to take issue with him or to offer any objections to the assertions he made in this paper. They are undoubtedly based on careful laboratory and clinical investigations, although how he knows whether a patient who comes into his office has a true cold or a false cold, whether the streptococci are latent or whether they have changed to Str. viridans, I do not know. If his theory is true, most of the people in the Mississippi Valley, noted for its sudden changes in temperature and humidity, have chronic hyperplastic sinusitis, and true cold, except, as Dr. Sewall defines it, that is, as an exacerbation of chronic sinusitis, is a rarity in this community.

I do not accept the theory that a cold may be produced without bacteria. The virus may be the symbiotic factor in arousing the latent bacteria in the nose—and there are always bacteria in the nose—to activity, along with certain biochemical changes due to changes of body temperature and fatigue.

Dr. Sewall has found the principal factor in colds (true colds or false colds), which is a change in body temperature, and consequently a biochemical change in the body which makes one less resistant to the bacteria present.

I should like to add to his theory that a cold is due to changing conditions of the body my own belief that fatigue in combination with chilling of the body, or

change in the body temperature, nearly always accompanies the onset of either the ordinary acute cold or the false cold described by Dr. Sewall.

I believe that the reason there is not more progress made in the matter of handling the common cold, or in preventing the epidemics of the common cold, is that not enough attention is paid to hygiene. The grandmothers of the present generation knew that if one got tired and sat in a draft, one would get a cold, and this is still so.

Dr. T. R. Gittins, Sioux City, Iowa: What I have to say in this discussion is based purely on clinical observations in private practice. I have no contact with research workers and am utterly incapable of discussing the author's remarks concerning the bacteriologic and pathologic aspects.

I have felt that the lessening of nasal infections as age advances is due to the increased general immunity developed by repeated contacts with various strains of the usual organisms encountered rather than to local tissue changes in the nasal and sinus mucosa.

Dr. Sewall stated that the altered mucosa secondary to allergic or biochemical changes is the forerunner of chronic sinusitis, which in turn is the source of the common cold. From a purely clinical standpoint, I must say that in every-day practice a patient with an allergic tendency is very resistant to infection, whether it is in the nasal or in the sinus membrane or elsewhere in the body—the appendix, the gallbladder, the skin, etc.

In speaking about the false cold, Dr. Sewall said: "Such vasomotor exacerbations may occur with greater or less frequency in those suffering from chronic hyperplastic sinusitis." I should add—or in those with an allergic tendency but without demonstrable hyperplastic sinusitis. It seems to me that the term chronic hyperplastic sinusitis has been overworked in the literature on otolaryngology and at times is used to designate an almost mythical condition, unless one also wishes to speak of the pale edematous nasal mucosa so commonly considered to indicate chronic hyperplastic rhinitis.

Again the following statement by the essayist stimulates discussion: "Without the ever replenished reservoir afforded by chronic sinusitis there would be no true common colds." I have felt that the gradually increasing incidence of acute nasal and sinus infections in the fall and early winter depends to a great extent on trauma to the nasal mucosa from improperly humidified air in heated buildings and from quick climatic changes. These alterations in the soil of the host probably allow the organisms which are already present on or in the nasal mucosa to become active. The presence of chronic sinus infection has not seemed necessary.

Dr. Howard C. Ballenger, Winnetka, Ill.: In general, I agree with Dr. Sewall's conception of the etiology of the common cold, especially in those persons who have an acute exacerbation every few weeks during the winter months. The great majority of these persons seem to have an underlying chronic sinusitis as the basis for their susceptibility to colds; in fact, these colds should be called acute exacerbations of chronic sinusitis. Many of these acute exacerbations are not of the vasomotor or allergic type but of the profusely discharging mucopurulent type, which can have only an infectious agent as the important causative factor. As stated by Dr. Sewall, chronic sinusitis probably furnishes the background for most of these acute phases. However, I fail to see why chronic tonsillitis or adenoiditis or infection of any other lymph structures could not act in a similar capacity. This opinion is also borne out in the laboratory by the demonstrable presence of the known pathogenic germs in the crypts or ducts of the lymphatic structures in many of these susceptible persons.

The work of Dochez and his associates in attributing the common cold to a virus which activates the known pathogens of the respiratory mucosa seems to be fairly well proved. The work in regard to influenza is not so clear. However, the same or a similar symbiosis may be present in persons with influenza as well as in those with the common cold. As a practicing otolaryngologist, one knows that from 80 to 90 per cent of the complications of the so-called common cold and influenza are due to hemolytic streptococci.

The question of pleomorphism, transmutation and dissociation of bacteria is a fascinating subject and offers interesting speculative possibilities. It explains in a satisfactory manner the relationship of the hemolytic streptococcus and the green-producing cocci. However, the subject is still in an embryonic stage, and definite conclusions cannot be reached as yet.

Chronic sinusitis is probably an important source of the streptococcus. However, I believe from clinical observation and from some bacteriologic investigation that other sources, such as chronic infection of the tonsil crypts or other lymphatic structures of the upper respiratory tract, may also act as a source. The known pathogenic bacteria in these various locations should be activated by the cold-producing virus the same as in the sinus.

I am not clear as to whether Dr. Sewall intends to convey the impression that a cold develops only in the presence of chronic sinusitis or altered sinus mucosa. I presume he does not, as the common cold is so universal and occurs at all ages, especially in the young, in whom changes in the sinus mucous membrane are not so common or advanced as in adults. As a result one would expect a smaller incidence of colds in children if sinusitis were the only etiologic factor, whereas the reverse is true.

Dr. Robert F. Ridpath, Philadelphia: I do not agree with the essayist that chronic sinusitis is essentially the only factor in the production of a common cold. Several factors are involved—susceptibility, heredity, the condition of the upper and the lower respiratory tract and of the intestinal tract and environment, such as movie theaters, churches and convention halls. Whether bacteria or a virus is the offender is still an unanswered question.

There are certain preliminary conditions, however, which have been found to be essential for the symptoms generally recognized as cold, coryza and rhinitis. To contract a cold there must be a shock to the system. The shock may be caused by damp, wet or cold feet and ankles or by the sudden chilling of other parts of the body. One fails to realize the tenderness and the susceptibility of the feet, as evidenced by the fact that though encased in stockings and shoes with soles of considerable thickness, nevertheless one is conscious, through all of this, of dampness, of an evenness of what one is in contact with when walking and of various materials with which one comes in contact, such as carpets, metal, wood, stone and cement, and also one is cognizant of the smallest particle which may be stepped on even though it may be paper, a hair or a needle. If the plantar fascia is thus endowed and is so sensitive to external objects, it is equally sensitive to elements or changes which produce systematic manifestations. A person may be fatigued or have intestinal intoxication or other conditions which lower body resistance, and the fact remains that the vasomotor disturbance results in congestion of the membrane of the nose.

At this period there is a mass condition of the mucous membrane either as a whole or of an ischemic nature. This is quickly followed by congestion, lacrimation, itching or a pricklish feeling in the nasal cavity, with slight conjunctivitis and other typical symptoms. It is my opinion that up to this time bacteria play

an important part in the problem but that at this time they play a decided part and become the factor of main importance in the length of time the infection lasts and its sequelae. This infection must be considered as a systemic infection and not as a local entity.

As our essayist has mentioned, weather plays its part, but I am inclined to consider humidity more important than temperature.

Feeling as I do and being one who is very susceptible to colds, or conditions which act exactly like them, I cannot agree with Dr. Sewall's view that one has definite immunization for six months or more. Having had three attacks this past winter, I am forced to believe that I passed through three infections.

Dr. Herman Semenov, Los Angeles: I have had the privilege of following Dr. Sewall's work. While many of his observations are theoretical, they have scientific foundations. Hyperplastic sinusitis is the most common type of sinusitis. It represents mesenchymal and epithelial proliferation as a result of irritation. Changes develop in any tissue when it is irritated. These changes are a healthy defense reaction and not necessarily abnormal just because the membrane is thickened.

After a purulent discharge has been present for twenty-five or thirty years, it may disappear and the patient may have no more symptoms. When the membrane which was infected is removed years later, it is found to be perfectly normal except that it is greatly thickened and consists of scar tissue. A patient with this type of infection may have had remissions and exacerbations of what he calls colds.

I think that Dr. Sewall attempted to point out two types of colds: (1) the epidemic type to which every one is susceptible, due to any one of the many types of bacteria and viruses, and (2) the common garden variety of coryza that some persons obtain when sitting in a damp, chilly place. This reaction is not always due to sinusitis but is frequently associated with it, and if it is looked on as a reaction on the part of the membranes to halt the further extension of bacteria by edema, by bringing about a hyperemia and by flushing the cavity with a free discharge of mucus, I think sinusitis is more than a mere infection. One will find bacteria in 95 per cent of all cases of chronic hyperplastic sinusitis, and in these cases one may consider a cold something like an allergic response—a complicated picture of infection, defense reactions and hypersensitivity.

Dr. Edward Cecil Sewall, San Francisco: The time limit prevented the reading of much of my paper. This led to some confusion in the discussion.

What I wish to convey is that the person with chronic sinusitis is the carrier of the common cold. Besides the false colds to which chilling and indiscretions subject him, he is capable of originating within himself, independent of outside contacts, a true, infectious common cold. This is due to the fact that the bacteria are lurking constantly in his tissues and gain potency when his immunity has expired, a true common cold resulting. He is then capable of imparting his cold to others, either by a virus or otherwise, whether they have chronic sinusitis or not. Thus do epidemics of the common cold appear during the colder seasons which fan into activity the many chronic infections of the sinuses. The person without chronic sinusitis takes "cold" only when the infection is carried to him by one with an acute cold. He never takes cold from chilling.

Dr. Ridpath lends authority to the prevalent opinion when he says that he has had two or three colds in as many months. I believe that he has failed to recognize the difference between the true cold and the false cold.

NEOSYNEPHRIN HYDROCHLORIDE IN OTOLARYNGOLOGY

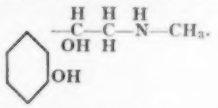
WILLIAM M. FITZHUGH JR., M.D.
PHILADELPHIA

It is the purpose of this paper to introduce to the otolaryngologist neosynephrin hydrochloride, a new vasoconstrictor which some pharmacologists believe to be superior to epinephrine and ephedrine for local intranasal application. Neosynephrin hydrochloride was accepted by the Council on Pharmacy and Chemistry of the American Medical Association about two years ago and is described in "New and Non-official Remedies," yet many otolaryngologists are not familiar with its biologic properties because investigators have reported their experimental and clinical results only in pharmacologic journals.

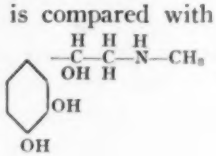
In order to make this paper complete I wish to give, first, a brief résumé of the chemistry of the drug and its relationship to other popular vasoconstrictors; second, a review of the previous biologic investigations and a report of the experiments on local intranasal application carried out for purposes of confirmation, and, finally, a résumé of fifteen cases, with a summary of the results of the application of neosynephrin hydrochloride in the Proetz displacement treatment for chronic sinusitis.

CHEMISTRY

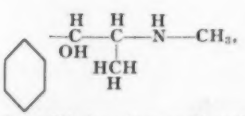
Neosynephrin hydrochloride may be described chemically as the hydrochloride of levo-metahydroxphenyl-1-methylamine-2-ethanol-1. It is also known as metasynephrin. The formula for the base is



When neosynephrin base is compared with the two more popular vasoconstrictors, epinephrine,



and ephedrine,



it will be immediately noticed that epinephrine has a second hydroxy radical in the para position and that ephedrine has no hydroxy radical

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on the benzene ring and has a second CH_3 group for a hydrogen atom on the side-chain. The similarity between these drugs in their chemical formulas does not necessarily mean that they must have identical actions, since there are other related compounds which have little or no effect on the animal organisms. There are also a dextro, a levo and a racemic compound, the levo compound being the form considered in this study. Neosynephrin is an isomer of parasynephrin.

Neosynephrin is a white crystalline substance, freely soluble in water as the hydrochloride. It is sufficiently stable so that an aqueous solution shows no apparent deterioration in strength after several weeks. It is oxidized, however, on coming in contact with metal; hence, solutions of the drug should be used only in atomizers without metal parts. In respect to stability, the solution may be boiled without decomposition; therefore, it resembles ephedrine rather than epinephrine.

PHARMACOLOGY

Pharmacologists are carrying out numerous investigations to find a perfect vasoconstrictor drug which has few or no toxic effects on therapeutic application. Neosynephrin hydrochloride is one of the newest synthetic compounds of this nature and appears in many respects to be much closer to this goal than epinephrine hydrochloride and ephedrine hydrochloride.

Circulatory Action.—Experimental work on animals has shown that neosynephrin hydrochloride is superior to both epinephrine hydrochloride and ephedrine hydrochloride. The intravenous injection into cats of 0.5 mg. per kilogram of body weight of the hydrochloride of the racemic mixture from which neosynephrin is made caused a rise in the blood pressure of 35 per cent, which was well sustained (lasting from ten to fifteen minutes), with little or no effect on the pulse rate and no diminution in the pressor action.

The administration to patients of therapeutic doses of the hydrochloride of the levo form, neosynephrin hydrochloride, by various routes showed a well marked rise in the systolic pressure, with a corresponding rise in the diastolic pressure. The latent period depended on the method of administration. The absence of general symptoms, even during the highest elevation of the blood pressure, should be reported here. "The most marked symptom observed was little more than an awareness of increased cardiac activity, so that for all practical purposes, it can be said that this drug was free from the unpleasant side-actions characteristic of ephedrine." [1]

Use in Infiltration Anesthesia.—If neosynephrin hydrochloride is a good vasoconstrictor it might be used as an adjuvant to local anesthetics

1. Personal communication from Dr. M. L. Tainter.

in infiltration anesthesia. However, it was found that epinephrine hydrochloride combined with procaine hydrochloride increased the efficiency of the anesthesia by sustaining its effects one-third longer than neosynephrin hydrochloride. The clinical value of neosynephrin hydrochloride for this use therefore requires further observation.

Effects on the Blood Sugar Content.—It has been known for some time that both epinephrine hydrochloride and ephedrine hydrochloride raise the blood sugar content and are of therapeutic value against hypoglycemia. Neosynephrin hydrochloride in therapeutic doses has only a slight effect in raising the blood sugar content.

Influence in Allergic Reactions.—Neosynephrin hydrochloride does not prevent anaphylactic shock in sensitized animals. From an early study it was found that this drug had little effect in diminishing the signs and symptoms of patients suffering from asthma or urticaria, while immediate relief followed the administration of epinephrine hydrochloride in appropriate doses. Later observations, however, indicated that neosynephrin hydrochloride is effective for certain allergic conditions.

Toxicity.—Neosynephrin hydrochloride has the greatest vasoconstrictor activity of any of the stable sympathomimetic amines; consequently it would be of interest to determine whether its toxicity increases proportionately to the pressor activity. This was determined experimentally on white rats, and it was found that neosynephrin hydrochloride had only one-eighth the toxicity of epinephrine hydrochloride. So far no comparison has been made with ephedrine hydrochloride.

TOPICAL APPLICATION IN THE HUMAN NOSE AND THROAT

Certain drugs have been used for local application in the nose and throat to shrink congested mucosa and facilitate drainage. Epinephrine hydrochloride has been used for this purpose for many years, but an increase in swelling is likely to follow the initial period of vasoconstriction. Cocaine hydrochloride relieves turgescence, but if used too often it may cause addiction, or there may be unpleasant and dangerous systemic reactions. Ephedrine hydrochloride has been used in the last few years for the same purpose, but it may also have several universal disadvantages, such as the production of pain and irritation, with secondary swelling and engorgement, besides occasional unpleasant systemic reactions. Neosynephrin hydrochloride is a more powerful vasoconstrictor than either epinephrine hydrochloride or ephedrine hydrochloride and does not cause susceptibility to addiction like cocaine hydrochloride; hence the question arose whether it would have a possible therapeutic field for shrinking turgescent or congested nasal mucosa.

Experiments Conducted by the Department of Otolaryngology, Stanford University.—These experiments were carried out in the Sewall

Clinic. A 0.5, a 1 and a 2 per cent solution of neosynephrin hydrochloride, diluted with physiologic solution of sodium chloride, were made up, with a 0.5 per cent solution of chlorbutanol as a preservative. These solutions were instilled or applied on a tampon into one nasal cavity, while the other nasal cavity was similarly treated with a 3 per cent solution of ephedrine hydrochloride as a control. When the 0.5 per cent concentration of neosynephrin hydrochloride was used good constriction occurred in two minutes which was indistinguishable from that caused by the 3 per cent solution of ephedrine hydrochloride. This constriction lasted two and one-half hours, or one-half hour longer than that caused by the ephedrine hydrochloride. When a 1 per cent solution was substituted for the 0.5 per cent solution the constriction was maximal in two minutes also, but it lasted for four hours, or twice as long as that produced by ephedrine hydrochloride. With these concentrations there were no signs of irritation, and the patient did not complain of any objectionable symptoms. However, when a 2 per cent solution was used the constriction was maximal in one-half minute, with a four hour duration, but there were signs of irritation. The department of otolaryngology of Stanford University reported no deleterious effects in the use of a 0.5 and a 1 per cent solution of neosynephrin hydrochloride and stated that in appropriate concentration the drug is superior to ephedrine hydrochloride in that no systemic or local reactions were noticed following its use.

Experiments Conducted in Philadelphia Hospitals.—These experiments were carried out in the Philadelphia General Hospital, the Graduate Hospital of the University of Pennsylvania, the Hospital of the Protestant Episcopal Church and the Rush Hospital. First it was necessary to verify the results of the department of otolaryngology of Stanford University, so I made up two solutions of neosynephrin hydrochloride, one a 0.5 per cent solution and the other a 1 per cent solution, diluted with physiologic solution of sodium chloride. These solutions were placed on tampons or applied with applicators to one nostril, while a 3 per cent solution of ephedrine hydrochloride, used as a control, was applied in the same manner to the other nostril. I then went a step further and used a 0.25 per cent dilution of neosynephrin hydrochloride, with a corresponding dilution of ephedrine hydrochloride as a control. The experiments and results were as follows:

1. In ten cases the 1 per cent solution of neosynephrin hydrochloride was applied with a tampon to a turgescent mucous membrane near the right middle meatuses and removed at the end of fifteen minutes. The average duration of the effect was three hours and forty-five minutes, while the effect of the 3 per cent solution of ephedrine hydrochloride in the left nostril lasted an average of two hours and fifteen minutes. In one of these cases there was marked local irritation with the control.

2. In twenty cases the congested mucous membrane was painted with the 1 per cent solution of neosynephrin hydrochloride on one side and with the 3 per cent control solution on the other. It was found that the average latent period before maximal shrinkage occurred was in the vicinity of two minutes for both solutions. Care was taken to see that there was no excess solution on the applicator prior to treatment.

3. Seven patients were given ½ ounce (14.2 cc.) of a 1 per cent solution of neosynephrin hydrochloride to be applied in divided doses every four hours to each nostril in the Beck position. Six of the patients reported no diminution of effect on each application, while the report of the seventh patient was unreliable —she had not followed instructions.

4. Four of the patients reported on in paragraph 3 were then given a 3 per cent solution of ephedrine hydrochloride to be applied in the same manner as in the former experiment. They admitted that the treatment with ephedrine hydrochloride was not as effective as the previous medication. One complained of a slight tremor, which lasted for fifteen minutes after every application. I tried the 1 per cent solution and then the control solution on myself in a subsequent attack of coryza, with the same results.

5. In sixteen cases tampons moistened with the 0.5 per cent solution of neosynephrin hydrochloride were used. It was found that the average time of constriction was two hours and forty-five minutes, or approximately half an hour longer than that produced by the 3 per cent solution of ephedrine hydrochloride.

6. In twenty-seven cases a 0.25 per cent solution of neosynephrin hydrochloride was applied with a tampon in one nostril and ephedrine hydrochloride of the same concentration was used in the second nostril. The tampons were removed in twenty minutes. The average duration of shrinkage for the neosynephrin hydrochloride was one hour and forty-five minutes, or almost double that for the ephedrine hydrochloride, which was about one hour.

7. Fifty patients were given ½ ounce of a 0.25 per cent solution of neosynephrin hydrochloride, to be applied in divided doses every three hours. After every application they all had relief from the symptoms. Two of these patients obtained no relief from the ephedrine hydrochloride, and a third had an attack of marked tremor, with palpitation, following the use of this drug.

Comment.—My investigation in a series of one hundred and thirty cases has shown that neosynephrin hydrochloride is superior to ephedrine hydrochloride in lengthening the duration of constriction of the mucosa, that it is less irritating and that there is a minimum of side-reactions on topical application.

USE OF NEOSYNEPHRIN HYDROCHLORIDE IN THE PROETZ DISPLACEMENT TREATMENT

The intranasal topical application of neosynephrin hydrochloride produces the desired shrinkage of turgescent mucous membrane without any marked local irritation or systemic reaction. Owing to the fact that the mucous membrane of the sinuses is continuous with that lining the nose, the question arose as to the advantage in substituting neosynephrin

hydrochloride for the 0.25 per cent solution of ephedrine hydrochloride in the Proetz displacement treatment.

It is not my intention to describe in detail the Proetz displacement treatment, which was so well covered by A. W. Proetz in his monograph on displacement, yet it is felt that a brief résumé of this form of therapy would not be out of order.

First, any purulent exudate or excess mucous should be removed before the displacement treatment is started. The patient is then placed in the supine position in a chair with his head resting on the lap of the operator, so that the chin and external auditory meatus are in a line perpendicular to the floor. The patient is then asked to repeat the letter "k" in rapid succession, while from 2 to 4 cc. of a 0.125 per cent solution of neosynephrin hydrochloride is instilled into one nostril, following which the alternating suction of not more than 5 pounds (2.3 Kg.) is applied to the same nostril, the operator holding the opposite nostril closed with the finger. The same procedure is then repeated for the other nostril, after which the patient is allowed to sit up. Rapid fluctuation in pressure has been carried out in all these cases, and it is felt to be as effective as the method employed by A. W. Proetz, viz., raising the negative pressure to 5 pounds twelve times. Often more solution is placed in each nostril, and an attempt is made by prolonging the treatment to irrigate the sinuses partially by allowing the medicament to wash them out.

RÉSUMÉ OF CASES

CASE 1.—G. K., aged 11 years, following tonsillectomy and adenoidectomy showed signs of inflammation of the posterior ethmoid sinus. The symptoms were manifested by hawking, and on oral examination one could see a postnasal mucopurulent discharge. When Proetz' solution (a 0.25 per cent solution of ephedrine sulfate in physiologic solution of sodium chloride) was used in the displacement treatment the nasal mucosa became secondarily congested; when neosynephrin (a 0.125 per cent solution in physiologic solution of sodium chloride) was substituted the condition cleared up after eleven treatments.

CASE 2.—H. W., aged 24, a teller in a bank, had severe intermittent headache in the frontal region for the past four or five years. This usually occurred in the morning, lasting from one to two hours, then abating. It was more frequent during stuffy and damp weather. The nasal mucosa was red, and the septum was thickened and deviated high up to the left, with small basal ridges on the right and the left. Infraction of both middle turbinated bones, the displacement treatment being combined with the use of the Proetz solution (a 0.25 per cent solution of ephedrine sulfate in physiologic solution of sodium chloride) gave decided benefit. There was a remission of symptoms after four such treatments, and the patient has been free from headaches for more than three months. The patient was not benefited by the use of neosynephrin hydrochloride, but the headaches disappeared when the regular solution of ephedrine hydrochloride was used.

CASE 3.—M. L., aged 43, a housewife, was referred to the clinic with a diagnosis of toxic neuritis of the optic nerve. When she was given the Proetz

treatment with neosynephrin hydrochloride her condition gradually improved. Later she is to have a submucous resection. This patient is still under treatment.

CASE 4.—A waitress aged 44 had headache in the frontal and the occipital region of many years' duration. There are a number of factors responsible in this case which are beyond control. The displacement treatment with the regular solution has helped appreciably, and the 0.125 per cent solution of neosynephrin hydrochloride has caused no appreciable biologic difference. The patient received in routine management many other modifications of the displacement treatment and hygienic guidance, so that her improvement cannot be ascribed to any one method of treatment.

CASE 5.—A. G., a housewife, had severe headache in the occipital region, especially during the menstrual periods. Cocainization of the sphenopalatine ganglion and of the "genital spots" of the nose helped. Neosynephrin hydrochloride was not superior clinically to the Proetz solution in this case.

CASE 6.—G. L., a physician aged 41, came to the clinic with the complaint of gradual progressive deafness and postnasal discharge. His eustachian tubes were treated by catheterization, and the chronic inflammation of the posterior ethmoid sinus was treated by displacement. The patient complained of a contracted urethral sphincter (slowness in starting the stream) and occasional palpitation when the solution of ephedrine hydrochloride was used, while no unpleasant effects occurred when neosynephrin hydrochloride was used. The period of benefit was no longer, however, with the 0.125 per cent solution of neosynephrin hydrochloride than with the 0.25 per cent solution of ephedrine hydrochloride.

CASE 7.—H. F., a bookkeeper, had large intumescent inferior turbinate bones. He complained of nasal obstruction, rhinorrhea and postnasal discharge. It was questionable whether there was an allergic background. Treatment consisted of cauterization of the inferior turbinate bones and, later, the Proetz displacement treatment. When used alternately with the regular solution of ephedrine hydrochloride, the solution of neosynephrin hydrochloride did not show any superiority. The patient's airway is from 40 to 60 per cent better than before the treatment.

CASE 8.—N. D., a lawyer, has occasional intense pain in the frontal region, which usually follows a neglected cold. Displacement treatments have not diminished the symptoms, regardless of the solution used. It appears that there is remission of the pain following bilateral maxillary irrigation. The washings are not grossly productive (only a few hurds) 90 per cent of the time.

CASE 9.—B. F., a milkman aged 39, had a swollen left eye with headache in the supra-orbital region, radiating to the vertex. On examination the left nasal passage was found to be filled with purulent exudate, which appeared to be coming from the olfactory cleft. The septum was deflected to the left and helped to block drainage. After displacement treatment with neosynephrin hydrochloride the patient showed marked improvement. He is going to have a submucous resection as soon as the weather permits.

CASE 10.—R. K., a housewife, had a pale-looking membrane, although all the symptoms were suggestive of inflammation. There was no history of allergy. The septum showed a sharp ridge on the left base and a high deviation to the right, with contact points. After submucous resection with trimming of the left middle turbinate bone, followed by further local treatment, there was decided improvement, which the patient estimated at from 75 to 80 per cent, though I believe that 30 per cent is more correct. In this case the solution of neo-

synephrin hydrochloride was more effective in the displacement treatment than the solution of ephedrine hydrochloride.

CASE 11.—M. B., a housewife aged 39, came to me complaining of postnasal discharge and headache in the region of the vertex, which was more severe two or three hours after she arose in the morning. The displacement treatment with neosynephrin hydrochloride appeared to clear the postnasal discharge, but the headaches continue. One of the middle turbinate bones is jammed against the septum; the patient will not have this condition corrected.

CASE 12.—R. S., a housewife, gave a history of swimming in the ocean in the fall of 1935, following which suppurative otitis media developed on the left side, with spontaneous rupture. The discharge continued for two months. Examination with a nasal pharyngoscope revealed evidence of inflammation of the posterior ethmoid sinus. The displacement treatment with either the solution of ephedrine hydrochloride or the solution of neosynephrin hydrochloride appeared to have no effect on the ethmoiditis. After the nasal mucosa was shrunk with 4 per cent solution of cocaine applied on tampons, followed by infra-red irradiation, all the symptoms gradually diminished.

CASE 13.—S. H., a bank clerk, gave a history of the frequent occurrence of colds and sore throat prior to tonsillectomy, following which he did not have sore throat but still suffered from colds. When he came to the clinic he complained of nasal obstruction and stuffiness in the ears, of two weeks' duration. Examination showed congested mucous membrane with a mucopurulent discharge. The nasal mucosa shrank well on treatment with a 1 per cent solution of neosynephrin hydrochloride applied on tampons, following which displacement treatment with a 0.125 per cent solution of metasynephrin hydrochloride was instituted. The patient said that he had immediate relief.

CASE 14.—C. C., a clerk aged 24, complained of intermittent headache in the frontal region, which sometimes radiated to the occiput. Various corrective measures had been instituted, i. e., submucous resection, operation on the middle turbinate bones to improve aeration, and drainage, without results. Shrinking with cocaine alone gave temporary relief, but when this was combined with Proetz displacement with either neosynephrin hydrochloride or ephedrine hydrochloride the period between exacerbations was prolonged.

CASE 15.—R. K., a clerk aged 27, complained of severe pain in the vertex. Examination of the nose showed purulent exudate in the olfactory cleft. On being questioned the patient admitted that he had a postnasal discharge with hawking. The condition steadily improved under displacement treatment with either neosynephrin hydrochloride or ephedrine hydrochloride, but the patient said that he had a slight feeling of apprehension with the ephedrine hydrochloride, which did not occur with the neosynephrin hydrochloride.

Comment.—My clinical results from the use of the Proetz treatments with the substitution of a 0.125 per cent solution of neosynephrin hydrochloride for Proetz' 0.25 per cent solution of ephedrine hydrochloride are rather confusing. Among the fifteen cases reported, it will be noticed that in five neosynephrin hydrochloride appeared to have no effect in abating the symptoms; in three the drug's action was similar to that of ephedrine hydrochloride, and in seven it appeared to give relief and was superior to the control solution. It must also be men-

tioned that the solution of neosynephrin hydrochloride (0.25 per cent) was one-half the concentration of the solution of ephedrine hydrochloride (0.25 per cent), which should be kept in mind when one compares the two drugs as to the amount and duration of constriction produced. During the period in which the displacement therapy was used, other treatments, so far as it was practicably possible, were discontinued.

There is no doubt that one has many handicaps to overcome in experimental work of this kind. I shall mention a few here:

First, the number of clinics containing vacuum machines are limited.

Second, as in my experience, the patients may be generally irresponsible and attend clinics only when they have an exacerbation of symptoms; consequently many of the data are indefinite and have to be discarded.

In order that the results in this paper may be regarded as conclusive, I believe that experimental work should be conducted on the effect of a 0.125 per cent solution of neosynephrin hydrochloride on ciliated epithelium, in addition to further study of clinical cases.

I also believe that although neosynephrin hydrochloride is somewhat more expensive than the solution of ephedrine hydrochloride, it can be used in those cases in which the patient is sensitive to ephedrine and also should be substituted intermittently for the regular Proetz solution in cases in which long periods of treatment are required.

SUMMARY

1. Neosynephrin is chemically related to epinephrine and ephedrine.

2. Neosynephrin hydrochloride is superior to epinephrine hydrochloride and ephedrine hydrochloride as a cardiovascular stimulant.

3. It is as good as, but not superior to, epinephrine hydrochloride in increasing the efficiency of infiltration anesthesia.

4. Neosynephrin hydrochloride has no effect on the blood sugar content, nor does it protect the animal from allergic reactions.

5. It is less toxic than epinephrine hydrochloride or ephedrine hydrochloride.

6. On topical application neosynephrin hydrochloride produces constriction of longer duration than that caused by ephedrine hydrochloride, and there are no apparent undesired effects.

7. From the fifteen cases reported in this paper, in which the Proetz displacement treatment was used for therapy, it would appear that a 0.125 per cent solution of neosynephrin hydrochloride does not prolong vasoconstriction any longer than a 0.25 per cent solution of ephedrine hydrochloride, but in certain cases it is less toxic.

Frederick Stearns & Co. furnished the levo neosynephrin used in this study.

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PATHOGENIC FUNGI

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The causal relation of fungi to the diseases of the ear, nose and throat has not received the consideration warranted by its importance and frequency. During a period of twelve months I have observed seventeen patients with verified fungous infection of the ear, nose, throat and respiratory tract. These patients were seen in the routine of a private practice. Continued interest and search for mycotic infections will certainly reveal an increasing number of cases of this nature.

HISTORICAL NOTE

The earliest study of mycology preceded that of bacteriology by many years. The invention of the microscope immediately opened up a hitherto unknown world—a world peopled by grubs, maggots and crawling things that fascinated the early microscopists.

Athanasius Kircher of Fulda was probably the first to use the microscope in the detection of disease. One of his publications, dated 1658, describes his experiments on the nature of putrefaction, and he reported the development of maggots and worms in decaying matter.

It is to Hooke that the mycologist must pay tribute. In the era when Leeuwenhoek was displaying his 247 microscopes and Malpighi was reporting his study of the red blood cells and the capillaries, Robert Hooke found time to describe a filamentous fungus that he saw in the yellow spots on rose leaves. Further discoveries of yeasts, molds and fungi were made during the next century. In 1839 Langenbeck discovered the first parasitic fungus observed in man in lesions of the disease known as thrush. During the same period Schoenlein described a fungus as the cause of favus, and Gruby and Eichelstedt reported their findings in tinea circinata and pityriasis versicolor and proved these conditions to be caused by fungous infections. The interest of microscopists and clinicians in the study of mycology was diverted in the latter part of the nineteenth century by the discoveries of Koch and Pasteur in relation to the bacterial etiology of disease. The science of bacteriology soon crowded out the search for fungi, which has recently been resumed.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc.

DEFINITION

Fungi are filamentous plants of simple cellular structure and belong to that division of the vegetable kingdom known as the thallophytes. They have a more complicated structure than the bacteria and differ from the higher plants by growing in irregular masses. The plant body does not become differentiated into roots, stems and leaves as do those of the higher plants. The algae, which also belong to the thallophytes, are provided with chlorophyll and so are able to synthesize their food from inorganic compounds with the aid of the energy of sunlight. Fungi, because of the absence of chlorophyll, must obtain their food from organic matter synthesized by other organisms.

Fungi are classified as saprophytic or as parasitic according to whether they obtain their food from a dead organic substance or from a living organism. Some parasitic species are facultative saprophytes, and some saprophytic species are facultative parasites. The saprophytic fungi are widely distributed in nature and include the common molds and mildews. Many parasitic species, especially of the rusts, smuts and mildews, cause great destruction of cultivated crops. The world-wide blights that have attacked chestnut trees and plants of other species are also caused by fungi.

CLASSIFICATION

The classification of fungi is still in a state of confusion. No better classification can be found at the present time than that adopted by Castellani.[1]

Thallophyta
- **Algae**
  - Chlorophyll present and able to build up nourishment from carbon dioxide in the air
- **Fungaceae**
  - No chlorophyll; derive nourishment from plant and animal matter
    - Schizomycetes: Unicellular bacteria, multiplying by fission
    - Eumycetes: Unicellular and pluricellular organisms or filamentous plants, dividing or multiplying by sexual or asexual spores
    - Myxomycetes: A vegetative body, multinucleate naked plasmodium; not found in man
    - Eumycetes
      - Hyphomycetes: Fungi imperfecti reproducing by free conidia; septate mycelium
      - Ascomycetes: Reproducing by ascospores; mycelium septate when present
      - Phycomycetes: Reproducing by zygospores; mycelium nonseptate in the vegetative stage
      - Basidiomycetes: Reproducing by basidiospores; septate mycelium

NOMENCLATURE

The language used by mycologists is confusing to the novice in the subject. A definition of some of the elementary terms used is helpful in the reading of reports of instances of fungous disease and in a study of the literature of the fungi.

The mycelium is the vegetative body of most types of fungi and is composed of a mass of filaments called hyphae. When the mycelium

1. Castellani, Aldo: Fungi and Fungous Diseases, Chicago, American Medical Association Press, 1927.

is not divided into cells by partitions, it is said to be continuous, or nonseptate. When it is divided into cells, it is said to be septate. Special branches of the hyphae bear spores. The simplest form of reproduction is by the formation of budlike outgrowths from the plant body, which are called thallospores, or blastospores. This is illustrated by the yeasts. Portions of the mycelium may be cut off by partitions and protoplasm inside gathered into a mass which has a thickened wall and is known as a chlamydospore. Under unfavorable conditions of growth, the protoplasm contracts and secretes a thick membrane; these resting cells are known as gemmae. Spores which are cut off from the tip of a hypha are known as conidia or conidiospores, and the hypha bearing them is a conidiophore. Conidia composed of one cell are described as simple, while those composed of two or more cells are called compound. Sexual spores are formed by the fusion of sexual elements known as gametes. Arthrospores are produced by segmentation of the mycelium and are also known as oidia. Ascospores are formed in a structure known as the ascus, the number of spores in the sac being limited to two, four or eight and constant for the particular species producing them.

EXAMINATION AND CULTIVATION OF FUNGI

Two methods are used in the examination of material for the presence of fungi, the direct examination of the material and the cultivation of the organism. The direct examination of the material may be made by the naked eye, with the aid of a hand lens or by means of the compound microscope. The material may be examined in its natural state or it may be stained. As a rule, a wet unstained specimen is the most satisfactory. Because pus cells, fibrin and exudate obscure the field, it will be found helpful to clear the specimen with a few drops of a 20 per cent solution of sodium hydroxide. Cornbleet [2] used an alcoholic solution of sodium sulfide to clear his specimens and called attention to the fact that certain fungi are dissolved by sodium hydroxide. For the examination of stained specimens the various aniline dyes are used. The Gram stain can be used for the majority of fungi, if one keeps in mind that these organisms have a tendency to overstain.

CULTIVATION OF FUNGI

The most widely used medium is that of Sabouraud, which has as its base peptone, crude maltose and agar. Shaw [3] made use of a medium composed of dextrose, tartaric acid and agar. He found that most

2. Cornbleet, T.: A Reagent for Demonstrating Fungi in Skin Scrapings and Hair, J. A. M. A. **95**:1743 (Dec. 6) 1930.

3. Shaw, F. W.: A Monilia from the Respiratory Tract, J. Lab. & Clin. Med. **12**:968 (July) 1927.

fungi will grow luxuriantly on this medium, while the growth of bacteria is completely retarded.

For the routine examination of specimens it has been found that the placing of the suspected material on a glass slide, with the addition of a few drops of alcoholic solution of sodium sulfide, and covering with a cover glass is satisfactory for a wet specimen. Gram's stain is used if a stained specimen is required. Shaw's medium is made use of when the material is to be cultivated. Modification of this procedure will be found necessary when the identity of the fungus is obscure.

A simplified routine, such as that described here, which may be carried out in the office is desirable. Material to be examined is placed on a slide and treated with a few drops of an alcoholic solution of sodium sulfide, as described by Cornbleet. After a few minutes the wet specimen is covered with a cover glass and examined with both the low power and the high power lens. More information can be obtained by the examination of a wet specimen in a shorter time by this method than by the use of a fixed stained specimen.

After examination of the wet specimen, an attempt at cultivation may be made. A supply of culture tubes containing slant preparations of Shaw's medium can be kept in the office. If the tube is plugged and corked, the medium will not dry out and may be kept in stock for an indefinite period. Growth of the ordinary fungi on this medium is rapid at incubator temperature, and in forty-eight hours identification of the organism is possible. Bacterial growth is greatly retarded on this medium. Examination of the colonies with a hand lens of 10 diameters magnification will show mycelium and spore-bearing hyphae. Staining of fixed specimens with the usual aniline dyes, Gram's stain or some of the silver salts will give additional information and will aid in the classification of the growth. For the classification of the various fungi, the mycologist makes use of more refined methods, including fermentation tests, inoculation of animals and cultivation on mediums having various p_H values.

Since this paper is concerned only with those pathogenic fungi that have been found to affect the ear, nose, throat and respiratory tract, a list of the conditions to be described is given here.

Moniliasis	Actinomycosis	Torulosis
Sporotrichosis	Coccidiosis	Aspergillosis
Blastomycosis		

MONILIASIS

Moniliasis is defined as a group of conditions involving the skin, mucous membranes and viscera and associated with the constant presence of Monilia in the lesions, secretions or exudates from the affected tissues. Monilia is a generic name, and included under it are a large number of organisms with certain morphologic characteristics which

place them in the general class of Hyphomycetes, or fungi imperfecti, and in the group of yeastlike fungi.

Stovall [4] described a study of over 200 cultures of yeastlike fungi isolated from patients with thrush, bronchitis, asthma, pneumonia, vaginitis and generalized infection. Among this group he encountered Oidium, Blastomyces, Endomyces, Torula, Mycoderma and Monilia. Of the 200 specimens obtained, Monilia was found in 150.

Direct examination of material from a lesion caused by Monilia shows the yeastlike bodies to be round or oval and about 2.5 microns in diameter, many showing buds at their ends. The organism stains with all the aniline dyes and is gram-positive. In addition to the yeastlike bodies, a fresh specimen will show a meshwork of mycelial threads, usually branched. Septums are rarely seen.

Inoculation of Sabouraud's or of Shaw's medium will produce a growth after seven days at from 25 to 35 C.

Many of the species composing the genus Monilia are widely distributed in nature and may be found growing on dead leaves, decaying fruit or decomposing wood. Their relation to disease was first reported by Langenbeck, in 1839. He found the parasite in the patches of thrush on the oral mucosa, the pharynx and the intestinal tract of a patient dying from typhus. It is believed that some members of the group which are found in human tissue as saprophytes may under certain conditions assume parasitic habits, become pathogenic and give rise to lesions of the skin, mucous membranes and viscera.

Thrush.—It is the lesions of the oral cavity in moniliasis that catch the interest of the laryngologist. In normal persons, fungi of the genus Monilia may be present in the oral and pharyngeal cavities without giving rise to lesions. Not infrequently, however, because of an impaired state of health of the host or perhaps owing to a deficient mechanism of immunity, the organism acquires parasitic habits, becomes pathogenic and excites biologic tissue reactions, with resultant characteristic lesions.

Such tissue reaction in the oral mucosa is not infrequently observed in infants and in children who are poorly nourished and of low vitality and less often in older persons in association with other infections or with diseases of a debilitating nature. Clinically the condition manifests itself in the white or cream-colored patches or plaques of varying size and shape in the mucous membrane of the tongue, gums, cheek, lips and pharynx. These plaques appear as false membranes and are readily removed, sometimes leaving a raw, bleeding surface. The condition is usually acute and responds quickly to local treatment. A chronic type

4. Stovall, W. D., and Bubolz, A.: Identification of Certain Funguses Pathogenic for Man, Am. J. Pub. Health **22**:493, 1932.

of this infection has been observed, in which there are leathery plaques on the cheeks and the gums.

Monilial Infection of the Bronchopulmonary System.—This condition was first observed by Castellani in Ceylon and reported in 1905. The first case reported in this country was that described by Boggs and Pincoffs in 1915. These authors stated recently that if they were reporting the case again they would classify the condition under sporotrichosis.

The relation of fungi present in pulmonary secretions to the underlying pulmonary disease is threefold, as pointed out by Hamman.[5] The fungus may be present in the bronchial secretions as a saprophyte and have nothing to do with the pulmonary lesion. It may have entered the lung as a secondary invader, but having gained a foothold, it may mingle with the etiologic agents of previously existing disease and cause profound alteration in the course of events. The fungus may be the primary and the sole infecting agent in the disease.

Castellani classified pulmonary moniliasis into three types: mild, intermediate and severe.

The patient with the mild type is little disturbed by his infection. There is a slight cough, with some mucopurulent expectoration but no blood. Fever is absent. The condition may subside, remain stationary or progress to a more severe type.

In the intermediate type all symptoms are more severe and may simulate those of incipient tuberculosis. The patient complains of dyspnea and severe cough. There is usually fever of a septic type. Hemoptysis may occur. The physical signs in the chest are those of chronic bronchitis.

The severe type of the disease is chronic in its course and usually terminates fatally. The pulmonary involvement may be unilateral or distributed throughout both lungs. Pleurisy is usually present. The clinical picture is that of advanced pulmonary tuberculosis characterized by cough, expectoration, hemoptysis, septic fever, night sweats and emaciation. Roentgen studies do not reveal a distinctive picture and show only a pathologic process similar in appearance to that in other types of pulmonary infection.

A positive diagnosis of pulmonary moniliasis is justified only by the constant finding of a pathogenic species of Monilia in the sputum. The pathogenicity of the organism and its causative relationship to the disease can be proved only when its intrapulmonary inoculation into rabbits results in the production of small pulmonary nodules, showing endothelial proliferation, giant cells and Monilia.

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5. Hamman, L.: Diseases of the Lungs, New York, Oxford University Press, 1927, vol. 2, p. 111.

Treatment.—The successful treatment of the acute type of oral and pharyngeal moniliasis depends entirely on cleanliness and the topical application of mercurochrome in a 2 per cent aqueous solution, gentian violet or a 2 per cent solution of salicylic acid in alcohol. In the chronic variety of thrush treatment gives unsatisfactory results. The condition often persists for years in spite of continued treatment with roentgen rays, chrysarobin and carbon dioxide snow. The treatment of mild pulmonary moniliasis is symptomatic. In the treatment of severe infections potassium iodide is used in large doses, up to 300 grains (19.44 Gm.) a day. Vaccine therapy has been used, with no great success. Insulin has been suggested, even for use when glycosuria is not present.

SPOROTRICHOSIS

Sporotrichosis is an acute or chronic disease caused by several species of fungi of the genus Sporotrichum. The disease process is granulomatous, and lesions are found in the skin, mucous membranes, bronchopulmonary system and other systems of the body. In this country the cases reported have to a great extent occurred in patients living in the Missouri valley. Infection in man may take place under various conditions and through a variety of agencies. Infected plants or vegetable matter carrying the organisms directly into an area of broken skin or mucous membrane constitute probably the most frequent source of clinical sporotrichosis in man. Foerster expressed the belief that the barberry bush is the source of the infection in many instances. Vegetables, flour, wines and decaying material about stables may harbor the parasite. The intact intestinal mucosa is permeable to Sporotrichum, as proved by Davis.

The active etiologic agent is a filamentous spore-bearing fungus of the genus Sporotrichum. The spores, which are about 5 microns long and 3 microns wide and pear shaped, appear situated along the filaments, usually mounted on short slight stems, and grouped at the ends or on the branches of mycelial threads. The spores multiply by a process of elongation and division and subsequent development of radiating mycelium. Growth on most mediums appears in five days. Examination of fresh material, an alcoholic solution of sodium sulfide being used to clear the specimen, shows the oval, cigar-shaped bodies, frequently lying within a large mononuclear leukocyte. A stained preparation from fresh material shows the organisms to be gram-negative. Stained specimens grown on artificial mediums are gram-negative. The mycelium and spores grown on artificial mediums are acid fast.

The lesion of sporotrichosis of the skin is usually granulomatous and multiple and is often chronic. The lesion tends to ulcerate, with a cuplike depression at the apex when it has become necrotic. The

mucous membranes not infrequently become involved, with lesions similar to those found in the skin. The conjunctiva and the nasal, lingual, pharyngeal, oral and laryngeal linings have all been reported as the site of lesions. Instances have been reported frequently of infection of the conjunctiva and of the lacrimal sac.

Albright [6] described a lesion of the nose and the nasal septum which had the clinical appearance of syphilis. It was later identified as a granulomatous lesion caused by sporotrichosis.

Forbus [7] in 1927 stated that pulmonary sporotrichosis is rare. The identity of the condition is most often confused because of the similarity of the symptoms to those of syphilis and tuberculosis. Bronchiectasis is often present.

Lesions of sporotrichosis, wherever situated, respond rapidly to iodine therapy. Iodine may be used in the form of a saturated solution of potassium iodide, with a daily dose of 200 grains (12.96 Gm.). Compound solution of iodine U. S. P. has been used. The intravenous administration of sodium iodide is an effective way of saturating the system of the patient with iodine.

BLASTOMYCOSIS

The term blastomycosis is used to describe a group of closely allied clinical conditions characterized by the presence of granulomatous lesions in the cutaneous type of the disorder and by pulmonary, osseous, meningeal and other visceral manifestations in the systemic type of the infection. Certain mucous membranes show lesions similar to those found in the skin.

The question of the number of species of offending organisms in this disease is still unanswered. Castellani expressed the view that there is a plurality of species of fungi causing blastomycosis. This opinion coincides with the observed clinical facts. The organisms as recovered from exudates are found to be practically similar in all instances, while on artificial mediums certain cultural and microscopic variations may be noted.

A fresh specimen treated with an alcoholic solution of sodium sulfide and examined with subdued light, shows the organism to be a round or oval, highly refractile body, from 5 to 20 microns in diameter, containing granules of various sizes and often vacuoles. These refractile bodies are sometimes surrounded by a hyaline capsule. Budding forms are seen, and pairs united in the form of a figure 8 can be observed.

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6. Albright, George C.: Sporotrichosis of the Nose, Laryngoscope **33**:875 (Nov.) 1923.

7. Forbus, M. D.: Pulmonary Sporotrichosis, Am. Rev. Tuberc. **16**:599 (Nov.) 1927.

The organisms stain irregularly and have a tendency to overstain with the ordinary aniline dyes. Within the tissues stained with hematoxylin and eosin the parasites may be seen within the giant cells as circular or oval bodies with granular or vacuolated protoplasm and frequent budding.

Blastomyces grows readily at incubator temperature on dextrose agar or on Shaw's medium. Hamburger reported that the strains examined by him tend to multiply by budding when grown in the incubator and to show aerial hyphae and a filamentous structure when grown at room temperature.

The cutaneous lesions resulting from infection with Blastomyces are usually ragged, superficial ulcers with soft granulating floors and a purulent discharge, with frequent formation of thick crusts. Surrounding each ulcer is a dark red, sometimes a purplish, zone.

Blastomycosis of the tongue has been reported by New and others. In the case described by New [8] there was a nodular tumor at the base of the tongue, filling the entire pharynx. The mass was hard.

Blastomycosis of the larynx has been reported. Chevalier Jackson [9] reported a short series of cases observed by him, and knowledge of these laryngeal lesions was brought up to date by his classic article. The initial symptoms complained of by all his patients suggested either laryngeal or pulmonary tuberculosis. In all cases there was a severe paroxysmal cough, at first nonproductive and later with expectoration of small amounts of sputum. Dyspnea was present in all instances. Jackson described the larynx on indirect examination as showing a chronically inflamed mucosa with a grayish, minutely nodular surface in some portions of the lesion, with a few isolated minute yellowish nodules. In each case there was in some portion of the lesion an appearance, such as is observed after the application of silver nitrate. Some of the membrane came away after the patient coughed, leaving a red, inflamed, eroded surface which was ulcerated in small spots. The diagnosis was verified in Jackson's cases by the finding of Blastomyces in the tissues, secretions or excretions.

Infection of the lungs with Blastomyces constitutes the most frequent form of the systemic type of the disease. In about 95 per cent of the cases reported some degree of pulmonary involvement was present.

Medlar [10] has drawn attention to the similarity of blastomycosis and tuberculosis. He emphasized the similarity of the morbid anatomic

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8. New, George B.: Blastomycosis of the Larynx, Ann. Otol., Rhin. & Laryng. **37**:240 (March) 1928.

9. Jackson, Chevalier: Blastomycosis of the Larynx, Arch. Otolaryng. **3**:99 (Feb.) 1926.

10. Medlar, E. M.: Pulmonary Blastomycosis, Am. J. Path. **3**:305 (July) 1927.

structure and the histopathologic changes produced in infections due to fungi and in those due to the tubercle bacillus. He pointed out that the variety of pathologic lesions produced in these two diseases depends largely on the balance between the virulence of the infecting agent and the resistance of the infected host. Miller's [11] studies of the reticulum of the lung have shown that its growth and transformation into collagenous tissue differ in no way in the tubercle of blastomycosis and in the tubercle of tuberculosis. If the presence of the special organism is eliminated from the picture, the lesion is the same in the two infections.

The course of the disease is chronic. Some patients have lived for seventeen years. In nine of ten cases in which there is pulmonary involvement the condition progresses to death within one year.

Treatment of the pulmonary complication has been with large doses of potassium iodide and with roentgen rays. The treatment has not been a success.

ACTINOMYCOSIS

Actinomycosis may be defined as a local or constitutional disease belonging in the class of infectious granuloma and caused by any one of several species of fungi of the genus Nocardia, by Actinomyces or by Streptothrix. Petruschky divided the filamentous organisms into four groups. These include Leptothrix, which appears as simple threads without branching, Cladothrix, the filaments of which show false branching, Streptothrix, the filaments of which show true branching, and the forms characterized by the presence in the tissues of sulfur granules in which the filaments have stellate arrangement and clubbed ends. This last group are classed as Actinomyces.

Hamman described the organism when found in the tissues as forming irregular, rounded greenish or yellow masses of tangled branches of mycelium. They vary in size from minute particles to masses from 1 to 2 mm. in diameter. The larger masses are visible to the naked eye and are the characteristic sulfur granules. The central portion of the mass is made up of dense, closely packed mycelium, from which branches project radially. These branches are capped by a club-shaped bulbous covering of homogeneous refractive material. On section the central portion shows a mass of tangled fibers, cells and amorphous débris, about which there is a dense network of interlacing filaments and at the periphery a scalloped margin of club-shaped branches arranged radially. These are not always present, and when they are absent the parasite cannot be definitely identified. The organism stains by Gram's method. It can be grown anaerobically on Shaw's

11. Miller, William Snow: The Reticulum of the Lung: Its Similarity in Blastomycosis to That in Tuberculosis, Am. J. Path. **3**:315 (July) 1927.

medium or on dextrose agar. Treatment of a fresh specimen with an alcoholic solution of sodium sulfide reveals the organisms as masses of interlacing mycelium made up of fine, nonseptate branching hyphae, without chlamydospores or conidia.

These parasites are to be found in dust, pollen or the chaff from grain stalks. The saprophytic members of the group, which at times acquire parasitic habits and become pathogenic, may be found in the soil. They have been found living in the alimentary canals of larval insects as well as in the tonsillar crypts and the gastro-intestinal tract in man. In a study of the infection in man in the United States, Sanford and Voelker [12] reported the anatomic distribution of actinomycosis as follows:

In nearly 60 per cent of the cases the infection occurred about the head and neck. In more than 18 per cent the lesions were abdominal, involving the wall or cavity, the intestines, the liver or the appendix.

The thoracic region was involved in almost 14 per cent of the cases, the chest wall and the lungs being the chief locations. Involvement of all other organs and systems has been reported, either as part of a generalized infection or as solitary lesions. Actinomycosis was first reported in the United States by John B. Murphy [13] in 1885. He reported two cases of actinomycotic infection of the jaw. Similar cases had been observed in the European countries a few years before the report by Murphy.

Lingual Actinomycosis.—Figi in 1925 reported a series of 49 cases of primary actinomycosis of the tongue. Additional instances of this condition have been reported since that date. Five per cent of all hogs slaughtered are said to have encapsulated actinomycotic nodules of the tongue. In man, the condition begins with the formation of a small deep-seated nodule in the body of the tongue, which at first is painless and causes no discomfort. After a few weeks, the lump begins to increase in size and to stretch the mucous membrane under which it lies. At that time the patient experiences some pain. At the same time the nodule begins to soften, and on its surface there appears a pale yellow area, which has a tendency to rupture spontaneously and to discharge a small amount of pus containing the characteristic sulfur granules. The rupture of the nodule results in the relief of the painful symptoms. A number of these small nodules go through the same process of painful swelling, softening and rupture, and the result is induration and thickening, with loss of mobility, so that protrusion of the tongue becomes

12. Sanford, Arthur H., and Voelker, Minna: Actinomycosis in the United States, Arch. Surg. **11**:807 (Dec.) 1925.

13. Murphy, John B.: Actinomycosis in the Human Subject, New York M. J. **41**:17 (Jan. 3) 1885.

impossible. In the absence of treatment, the infection spreads to the surrounding soft tissues, with ulceration, glandular involvement and the formation of sinuses.

Pulmonary Actinomycosis.—Christison and Warwick [14] classified the pulmonary lesions of this disease into four groups:

Bronchitic Type: Infection is confined to the bronchi and characterized by the presence of pus and fungi.

Pneumonic Type: The process is spread out from the bronchi and the surrounding alveoli, which are filled with pus or rarely with fibrin. The lesions resemble those of ordinary bronchopneumonia.

Pleural Pneumonic Type: This is the pneumonic type, which has not organized and has resulted in the breaking down of tissue and in the formation of abscesses. In the pleural pneumonic type the abscesses have grown larger and have extended to the pleural cavity or even to the chest wall.

Metastatic Nodules: These are supposed to be carried by the blood stream and are considered to have no relationship to the bronchi.

The clinical history of pulmonary actinomycosis is in every way similar to pulmonary suppuration from other causes. The possibility of actinomycosis of the lung must always be considered when a patient is examined for pulmonary suppuration of a chronic type or for a condition resembling pulmonary tuberculosis save for the absence of tubercle bacilli in the sputum. In a recent case, not reported, the first complaint on the part of the patient was of constant pain in the right side of the chest. Two weeks after the onset of the pain there was a profuse expectoration of material of a foul nature. Four weeks after the onset of pain in the chest there was blood mixed with the expectorated material. At that time sulfur granules and actinomycetes were found in the sputum.

The formation of sinuses through the chest wall was said by Lord [15] to be more commonly associated with actinomycosis than with any other disease causing pulmonary suppuration. He stated that it was reported in 82 of 100 cases collected from the literature. Another characteristic feature of the disease is the dense infiltration of the involved tissues, which become boardlike.

The bronchoscopic appearance of an actinomycotic bronchial tree is about the same as that in any other chronic pulmonary suppuration. The affected bronchi are reduced in size, become fixed and contain granulation tissue that bleeds freely on manipulation. Sections of this tissue show the ray fungus in great numbers.

14. Christison, J. T., and Warwick, M.: Actinomycosis of Lungs and Suprarenals, J. A. M. A. **89**:1043 (Sept. 24) 1927.

15. Lord, F. T.: Diagnosis in Case of Primary Pulmonary Actinomycosis, M. Clin. North America **8**:1485, 1925.

Actinomycosis of the Nose.—Actinomycosis of the nose is a not uncommon condition in patients living in regions in which actinomycotic infections are prevalent. The infection in the nose manifests itself as a granuloma and is similar in its gross appearance to the granuloma of the nose caused by tuberculosis, syphilis or lupus.

A granuloma of the nose caused by actinomycetes may be found on the septum or on one of the turbinate bones. Actinomycotic granulomas have been reported as involving the nasal sinuses. When found in the nares, the granulomatous mass is usually deep red, bleeds freely on manipulation and sometimes shows an ulcerated surface. Diagnosis can be made only by the finding of the offending fungus.

In a case observed by me the lesion was first thought to be a tuberculous granuloma because of the numerous giant cells seen in the section made from the tissue removed from the nose. The granuloma returned in a few weeks despite thorough removal and cauterization of the base. The appearance of yellow granules on the surface of the tumor led to the examination of the discharge and the finding of actinomycetes in great numbers.

ASPERGILLOSIS

Aspergillosis is an inflammatory disease caused by an infection with one of the species of fungi of the genus Aspergillus and of the family Aspergillaceae. The parasites have a special affinity for the pulmonary system and frequently attack the canal of the ear, the middle ear, the nasal sinuses and the conjunctiva. The inflammatory process is granulomatous in character, and the pathologic picture is similar to that found in the other infectious granulomas.

The members of the genus Aspergillus are widely distributed in nature. They are often found on dead or dying plant tissue. Tom and Church reported that they are abundant on ripe fruit, such as figs, dates and berries. The parasitic members of the group are comparatively few. They are frequently found as harmless invaders of the external auditory canal, the nasal sinuses and the external genitalia. They are found as secondary invaders in certain types of pulmonary suppuration.

Most members of the genus Aspergillus are saprophytic and have never been known to cause disease in man or in animals. About sixty members of the genus have been suspected of pathogenicity. Of this number, Aspergillus niger and Aspergillus fumigatus are the ones most commonly found.

The reproduction organ of Aspergillus is composed of a stalk and a spore-bearing head. The stalk, or conidiophore, has its origin in a foot cell in the mycelium. In some species a number of foot cells become connected to form long hyphae bearing many short stalks. The average diameter of the individual spores seen in the parasitic

aspergilli is from 2.5 to 5 microns, while the diameter of the fructification is from 20 to 30 microns.

Examination of fresh material is carried out by treating the specimen with an alcoholic solution of sodium sulfide. A preparation of this sort may be examined at once. Aspergilli are readily grown on all the laboratory mediums. A rapid growth is obtained on Shaw's medium at incubator temperature. When infected material, such as sputum, discharge from the canal or the middle ear or washings from the nasal sinuses, containing aspergilli is planted on the medium suggested by Shaw and incubated at 35 C., there appear at the end of twenty-four hours round white points, which rapidly increase in size and show a tendency to coalesce to form large plaques and to adhere to the medium. The color of the growth changes at once and varies greatly, depending on the species being cultured. Aspergillus fumigatus assumes a greenish tinge at first and after ten days takes on a smoky appearance. Aspergillus niger soon shows the surface of the medium covered with black granules.

Aspergilli are readily stained by the aniline dyes. The spores of most of the species are gram-positive, and the hyphae are gram-negative. In the tissues, the organisms lose their characteristics and may be observed only as mycelial threads or as rounded yeastlike bodies. The usual fructification is absent in the tissues.

Infection of the ear with Aspergillus has been recognized for many years. At the time of writing it is believed that certain members of the genus Aspergillus are capable, under favorable conditions, of initiating or prolonging an inflammatory process in the ear. This applies to the canal of the external ear as well as to the cavity of the middle ear.

The organisms found associated with this condition most frequently belong to the species A. niger, less frequently to the species Aspergillus fumigatus and rarely to the genus Mucor or Penicillium. The causative relationship of the last-named groups has been seriously questioned.

Infection of the external canal of the ear with Aspergillus may exist without symptoms for a long period. Itching of the ear is the most common symptom and may be intolerable to the patient. There is frequently a sense of fulness in the ear, with tinnitus. Sometimes furunculosis of the canal is present, probably due to a secondary infection of the tissues and of the broken epithelium with some of the pyogenic organisms. The drum membrane is usually involved in the process and is found to be deeply injected, with a loss of detailed markings. Hearing is reduced because of the mechanical obstruction to the sound waves by the collected discharge and débris. The canal is found partly filled with a mass of moist, foul material, which often has the

appearance of wet blotting paper. If the infection is due to A. niger, the material in the canal will be spotted with black granules.

While the usual picture is that given, it is found sometimes that the canal of the ear is dry and that the lining membrane is covered with dry scales and flakes. The condition has frequently been classified as eczema of the canal, and it is only after Aspergillus is found that the correct classification can be given to the infection.

Infection of the ear with Aspergillus is not confined to the external canal. A routine examination of the discharges from the middle ear through a perforation in the drum membrane will show a moderate number of instances of infection with members of the genus Aspergillus. It is possible that the presence of aspergilli in the discharge from what has been considered chronic otitis media may be due to the secondary invasion by aspergilli that are only saprophytes and have no causal relationship to the infection of the middle ear. It is a clinical fact that in some instances of infection of the middle ear in which the presence of aspergilli can be shown by direct examination and by cultivation on laboratory mediums, immediate improvement will occur if treatment is directed toward the control of the growth of the fungi.

There is nothing characteristic about the history or appearance of an infection of the middle ear caused by aspergilli. In most cases the discharge is of prolonged duration. The infection frequently shows remission, with a cessation of the discharge. A routine examination for fungi of all discharges from the middle ear and of all collections of scales, débris and moist masses from the canal of the ear will reveal a number of fungous conditions.

Aspergillosis of the nasal sinuses has been recognized as a clinical possibility for a number of years. Isolated cases have been reported by a number of trained observers. Holland Stevenson has recently reported seven instances of infection with Aspergillus fumigatus. Stevenson [16] in all seven of his cases assigned the infection to Mucor Histoides.

The clinical course of fungous infection of the nasal sinuses differs in no way from infections of these parts by the ordinary pyogenic organisms. It is only by the routine examination for fungi of the washings or discharges from the sinuses that the presence of fungi can be determined. Adams [17] has described an instance of infection of the ethmoid, maxillary and sphenoid sinuses in which A. fumigatus was recovered from all these parts. In all the cases reported the con-

16. Stevenson, H. N.: Mycotic Infections of the Maxillary Sinuses, Arch. Otolaryng. **20**:340 (Sept.) 1934.

17. Adams, N. F., Jr.: Infection Involving the Ethmoid, Maxillary and Sphenoid Sinuses and the Orbit Due to Aspergillus Fumigatus: Report of Case, Arch. Surg. **26**:999 (June) 1933.

dition has been chronic, has shown recurrence and has responded to the treatment used for more common types of sinus infection.

A routine examination of washings from the antrum and of discharges from the other sinuses would seem to be the only method by which a possible fungous infection of the nasal sinuses can be discovered. A direct examination of the material, using an alcoholic solution of sodium sulfide for clearing the specimen of blood, fibrin and pus cells, will show the organisms in a limited number of cases. Cultivation of the suspected material on the medium suggested by Shaw will reveal some additional instances of this infection.

Aspergillosis of the Tongue.—Douglas Macfarlan [18] reported a case of black tongue in a young person with no other demonstrable disease. Examination of scrapings from the affected area by staining and cultivation showed the presence of Aspergillus niger. His treatment, not entirely empirical, was to swab the tongue with glycerite of tannic acid, followed immediately by the application of silver nitrate, 25 per cent strength. The surface of the tongue became a deeper black, which was followed in a few days by a complete clearing of the organ. There was no recurrence of the condition.

I observed a somewhat similar instance of black tongue in which no aspergilli could be found. A filamentous organism with spores was observed and cultivated but could not be classified. Treatment similar to that suggested by Macfarlan was used and resulted in disappearance of the heavy, black tufts in four days. There was no recurrence. Further attempts to classify the fungi in the original growth have been without success.

Bronchopulmonary aspergillosis was first observed and recognized in 1842. A. fumigatus is the parasite usually present in this condition. It is a not uncommon condition in birds, especially pigeons, and has been known to occur in domestic animals. In the human being, the organism is frequently a secondary invader in pulmonary tuberculosis and other suppurative conditions of the lungs. When the disease occurs as a primary infection of the lungs, it resembles chronic pulmonary tuberculosis. Fibrosis is common, and the formation of cavities is the rule. The typical lesion of aspergillosis in the lung is an area of necrosis surrounded by a zone of active inflammation. The necrotic portion becomes sharply demarcated from the uninfected portion of the lung and is coughed up, leaving a cavity. Because the organism is a secondary invader more often than it is the primary cause of a pulmonary lesion, great care must be used in diagnosing a condition as pulmonary aspergillosis on the strength of finding the organisms in the sputum.

18. Macfarlan, Douglas: Fungus on the Tongue, Laryngoscope **34**:720 (Sept.) 1924.

Schneider [19] stated that the signs and symptoms closely resemble those of pulmonary tuberculosis. There are usually anorexia, loss of strength, cough and an evening rise of temperature. Hemoptysis is frequent. Other fungi closely related to the aspergilli are sometimes found as secondary invaders in a tuberculous lung. Among these Penicillium and Mucor are the most frequent. Both these fungi are probably saprophytic.

The prognosis in pulmonary aspergillosis is generally bad. Instances of spontaneous recovery have been reported.

The treatment of aspergillosis of the aural canal and the middle ear always gives satisfactory results. The general rule of keeping the canal dry may be helped by the use of dusting powder containing iodine. The use of a 2 per cent solution of salicylic acid in 70 per cent alcohol dropped into the canal of the ear twice a day and allowed to remain for five minutes will clear up most infections of the canal and of the drum membrane. After the use of these drops for a few days, there will be desquamation of the epithelium of the membrane of the canal. The infection has a tendency to recur; so the patient is instructed to return to the use of the drops for a period of three days on the first indication of a return of the infection. A recurrence will be indicated by itching of the ear or by a sense of fulness in the canal of the ear.

Aspergillosis of the nasal sinuses is treated in the same manner as sinusitis caused by one of the more common organisms. The internal administration of iodine in the form of potassium iodide or compound solution of iodine U. S. P. has been used with no marked improvement. Pulmonary aspergillosis is a chronic infection for which treatment has been unsatisfactory. Huge doses of potassium iodide are always given. The intravenous administration of sodium iodide has been used. Radiation with high frequency roentgen rays has not been satisfactory.

OTHER FUNGOUS DISEASES

A number of other fungi have been found to be parasitic in man. Among these, note should be made of Coccidioides immitis, which produces a granulomatous disease termed coccidioidal granuloma. A second disease condition similar to this, termed torulosis and caused by a yeastlike parasite, Torula histolytica, is also classed with the infectious granulomas.

Both these organisms can and frequently do produce an infection of the pulmonary system. The changes produced in pulmonary tissue are similar to those caused by tuberculosis, by actinomycosis and by aspergillosis.

19. Schneider, L. V.: Primary Pulmonary Aspergillosis, Am. Rev. Tuberc. **22**:267, 1930.

There is a suggested significance about the close similarity between the clinical picture of tuberculosis of the lung and that seen in mycotic pulmonary infections. This resemblance between the two types of infection is carried out in the pathologic changes in the tissues and the histologic appearance in the two conditions. The similarity in the clinical course, roentgen findings and histologic changes observed in the infectious granulomas of the lung and infection with the tubercle bacillus suggests the thought that the tubercle bacillus may be a yeastlike organism at some time during its life cycle.

The reported experiments of Stephen Maher [20] on the mutations and progeny of the tubercle bacillus, with special reference to the yeastlike bodies, confirm this view of the fungous characteristics of the tubercle bacillus.

The Sartorys and Meyer,[21] in recent experiments with the tubercle bacillus grown on special mediums, found, in the third generation, a loss of the acido-alcoholic property. The bacilli at this stage take on the form of branching filaments, endowed with a pronounced power of longitudinal division and growth.

These authors found that there is a morphologic modification of the tubercle bacillus which shows evolutionary forms, confirming the work done by Roman and tending to show that the tubercle bacillus is more closely related to the fungi than to the bacilli and could reasonably be classified among the actinomycetes.

A final diagnosis of pulmonary tuberculosis, in the absence of the tubercle bacillus, should not be made in an instance of infection of the lungs until a search has been made for parasitic fungi in the expectorated material. This search must include cultivation of the suspected material on various mediums, under both aerobic and anaerobic conditions. It should include the intratracheal inoculation of laboratory animals with the organisms isolated.

A routine examination of sputum from patients in institutions for the care of the tuberculous will show the presence of fungi in a percentage of the specimens examined. Not all these fungi are parasitic. The presence of some of them in the lungs certainly has some relation to the course of the tuberculous infection.

While it is impracticable to examine the discharge from an abscess of the middle ear as a routine procedure, if such a procedure is carried out, the otologist will often be rewarded by the finding of fungi associated with chronic or recurrent infection of the canal or the middle

20. Maher, Stephen J.: The Progeny of the Tubercle Bacillus, Am. Rev. Tuberc. **31**:350 (March) 1935.

21. Sartory, A.; Sartory, R., and Meyer, J.: Mutations in the Tubercle Bacillus Under the Influence of a Tuberculin-Sulphoguaiacolic Medium, J. State Med. **43**:303 (May) 1935.

ear. The same suggestion is made in regard to the washings obtained by lavage of the nasal sinuses.

The reason for including the pulmonary manifestations of fungous infections in this survey is that the bronchoscopist is now brought in to aid in the diagnosis of many instances of pulmonary infection.

Bronchoscopy is a definite help in the diagnosis in instances of pulmonary suppuration from undetermined cause. It is possible by direct examination of the bronchial tree to determine the presence of granulation tissue in some of the infectious granulomas. In this granulation tissue fungi may be found. Secretions uncontaminated by bacteria from the mouth may be obtained from the finer divisions of the bronchi, and the material may be examined for organisms, including the fungi.

The bronchoscopist will find no characteristic picture in the instances of pulmonary suppuration due to infection with fungi. He will be able to describe the appearance of the bronchial tree and to obtain tissue and secretions, examination of which will be an aid in the final classification of the disease.

SUMMARY

Fungous infections of the ear, nose, throat and respiratory tract are being observed and reported in increasing numbers.

A routine is suggested for the simple, rapid examination of material harboring the more common fungi.

The similarity between the pulmonary lesions produced by the tubercle bacillus and by some of the pathogenic fungi is noted. The possibility is suggested that the tubercle bacillus may be a yeastlike organism in some phase of its life cycle.

A survey is made of the literature describing the fungous infections of the ear, nose, throat and respiratory tract.

CHOLESTEATOMA VERUM TYMPANI

ITS RELATIONSHIP TO THE FIRST EPIBRANCHIAL PLACODE

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Cholesteatoma of the middle ear and of the mastoid may arise either primarily as a congenital growth or secondarily as a result of chronic infection. The pathologic end-product, however, is the same, and it is somewhat confusing that two entirely different processes should be called by the same term. In this discussion the secondary type will receive no attention.

True, primary, or congenital, cholesteatoma must be classed among the rare types of tumor, but its occurrence in many parts of the body makes it an object of great interest. It has been noted in the breast, diaphragm, liver, gallbladder, stomach, intestinal tract, pancreas, spleen, kidney, urinary bladder, ovary, testis, uterus and other parts of the body.

However, its most common occurrence is in the head. Several authors [1] have reported cases in which the lower jaw was involved, and in 2 cases [2] the tumor lay in the floor of the mouth. Kyle [3] noted a tumor in the lateral pharynx and Wingrave [4] one in the supratonsillar fossa. The superior maxilla and the antrum have been the seat of a tumor in several cases,[5] and Otrich [6] reported a case in which the ethmoid sinus and the antrum were converted into one cavity by pressure necrosis.

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1. (*a*) Dupuytren, G.: Bull. de l'Ecole de méd. de Paris **3**:32, 1807. (*b*) Mikulicz, J.: Beitrag zur Genese der Dermoide am Kopfe, Wien. med. Wchnschr. **26**:953 (Sept. 23) 1876. (*c*) Kostecka, F.: Ein Cholesteatom im Unterkiefer, Ztschr. f. Stomatol. **27**:1102 (Dec.) 1929.

2. Bose, quoted by Marchand, F.: Beiträge zur Kenntnis der Dermoid-Geschwülste, Ber. d. oberhess. Gesellsch. f. Nat.- u. Heilk. **22**:325, 1883. de Rezende, M. O.: Um caso ravo de cholesteatoma do soalho da bocca, Rev. oto-laring. de São Paulo **1**:46 (Jan.-Feb.) 1933.

3. Kyle, J. J.: Cholesteatoma of Lateral Pharynx, J. Laryng., Rhin. & Otōl. **33**:366 (Dec.) 1918.

4. Wingrave, W.: A Cystic Cholesteatoma from the Left Supratonsillar Fossa, J. Laryng., Rhin. & Otol. **18**:208 (April) 1903.

5. Bremer, G.: Ueber Cholesteatom in der Highmorshöhle, Hygiea **89**:577, 1927. Winckler, E.: Zur Kasuistik des Kieferhöhlen-Cholesteatoms, Ztschr. f. Laryng., Rhin., Otol. **2**:251, 1910. Aronson, W.: Zur Frage der Cholesteatome der Kiefer, Ztschr. f. Stomatol. **32**:571, 1934.

6. Otrich, G. C.: Cholesteatoma Involving the Ethmoidal Cells and the Antrum of Highmore, Illinois M. J. **48**:397 (Nov.) 1925.

Even the orbit [7] and the iris [8] have given rise to cholesteatoma. Volkmann [9] reported a case in which the entire scalp was involved.

A fairly large number of cholesteatomas have been found in the frontal sinus,[10] the frontal bone,[11] the parietal bone,[12] the temporal

7. (*a*) Rohmer: Cholestéatome de l'orbite, Bull. et mém. Soc. franç. d'opht., 1889; cited by Rintelen.[7f] (*b*) Schirmer, O.: Ein Fall von Cholesteatom der Orbita, Beitr. z. Augenh. **4**:310, 1899. (*c*) Birch-Hirschfeld, A.: Die Krankheiten der Orbita, in von Graefe, A., and Saemisch, E.: Handbuch der gesamten Augenheilkunde, ed. 2, Berlin, Julius Springer, 1915, vol. 9, pt. 2, chap. 13, p. 556. (*d*) Starcke, H.: Zur Genese der Cholesteatome des Gesichtsschädels, Ztschr. f. Laryng., Rhin., Otol. **11**:189, 1922. (*e*) Michail, D.: Cholestéatome de l'orbite, Arch. d'opht. **48**:743 (Nov.) 1931. (*f*) Rintelen, F.: Zur Kenntnis der Dermoidzysten und Cholesteatome der Orbita, Klin. Monatsbl. f. Augenh. **92**:165 (Feb.) 1934.

8. Rothmund, A.: Ueber Cysten der Regenbogenhaut, Klin. Monatsbl. f. Augenh. **10**:189, 1872. Monoyer, F.: Epithélioma perle ou margaritoide de l'iris, Gaz. méd. de Strasbourg **32**:3 (June 1) 1872.

9. Volkmann, R.: Cholesteatom der Kopfschwarte, Arch. f. path. Anat. u. Physiol. **13**:46, 1858.

10. Gilibert, J. E.: Adversaria medico-practica prima, Lugduni, J. B. Delamolliere, 1791, p. 27; quoted by Steiner, F.: Ueber die Entwickelung der Stirnhöhlen und deren krankhafte Erweiterung durch Ansammlung von Flüssigkeiten, Arch. f. klin. Chir. **13**:181, 1872. Weinlechner: Demonstration eines durch Trepanation geheilten Cholesteatoms der Stirnhöhle, Wien. klin. Wchnschr. **2**:136, 1889. Kahler, O.: Zur Frage der Genese der Cholesteatome der Nebenhöhlen, Wien. med. Wchnschr. **21**:562 (April 16) 1908. Hald, P. T.: Fall von Sinusitis frontalis Cholesteatomatosa (mit Demonstration), Internat. Zentralbl. f. Ohrenh. **7**:357, 1909. Wolff, H.: Ueber ein Cholesteatom der Stirnhöhle, Beitr. z. klin. Chir. **130**:215, 1923. Spencer, F. R.: Primary Cholesteatoma of the Sinuses and Orbit: Report of a Case of Many Years' Duration Followed by Carcinoma and Death, Arch. Otolaryng. **12**:44 (July) 1930.

11. (*a*) Esmarch, F.: Klinische Beiträge: I. Cholesteatom im Stirnbein, Virchows Arch. f. path. Anat. **10**:307, 1856. (*b*) Wotruba, C.: Ueber ein Cholesteatom im Stirnbein, Wien. klin. Wchnschr. **2**:899 (Nov. 21) 1889. (*c*) Marx, H.: Ein echtes Cholesteatom des Stirnbeins, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **23**:273, 1926. (*d*) Kahler, O.: Zur Kenntnis der primären Cholesteatome des Stirnbeins, Ztschr. f. Laryng., Rhin., Otol. **23**:227, 1932. (*e*) Loepp, W.: Die Cholesteatome des Schädels, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **138**:65, 1934. (*f*) Bucy, P. C.: Intradiploic Epidermoid (Cholesteatoma) of the Skull, Arch. Surg. **31**:190 (Aug.) 1935. (*g*) Lauterburg, W.: Ein Epidermoid frei im Wirbelkanal und seine Kombination mit Hirnläsionen, Virchows Arch. f. path. Anat. **240**:328, 1923.

12. (*a*) Blecher: Ueber Cholesteatom (Epidermoid) der Schädelknochen, Deutsche Ztschr. f. Chir. **70**:352, 1903. (*b*) Orlandi, N.: Ueber ein echtes Cholesteatom des Scheitelbeins, Virchows Arch. f. path. Anat. **237**:119, 1922. (*c*) Cushing, H.: A Large Epidermal Cholesteatoma of the Parieto-Temporal Region Deforming the Left Hemisphere Without Cerebral Symptoms, Surg., Gynec. & Obst. **34**:557 (May) 1922. (*d*) Kempmann, W.: Das Cholesteatom des Schädeldachs und seine entwicklungsmechanische Differenz im Vergleich zu

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squama,[13] the sphenoid[14] and the occipital bone.[15] In several cases[16] the occipital bone was destroyed although the tumor apparently arose from the dura. A similar case in which the posterior surface of the petrous bone was involved was reported by Berger.[17] Giraldes[18] reported a cholesteatoma of the anterior fontanel in an infant, and Bucy[11f] noted a tumor of the same region in an adult.

Although this tumor has been observed in various portions of the brain, as the frontal region[19] the corpus callosum,[20] the base of brain,[21]

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den basalen Perlgeschwülsten, Beitr. z. klin. Chir. **139**:343, 1927. (*e*) King, J. E. J.: Personal communication, cited by Horrax, Yorshis and Lavine.[21o] (*f*) Bucy.[11f] (*g*) Critchley, M., and Ferguson, F. R.: The Cerebrospinal Epidermoids (Cholesteatoma), Brain **51**:334 (Oct.) 1928.

13. (*a*) Virchow, R.: Ueber Perlgeschwülste (Cholesteatoma Joh. Müllers), Virchows Arch. f. path. Anat. **8**:371, 1855. (*b*) Schwartze, H.: Cholesteatoma verum squamae ossis temporum, Arch. f. Ohrenh. **41**:207, 1896. (*c*) Beyer: Cholesteatoma Verum, ibid. **91**:162, 1913.

14. Unterberger, F.: Ein echtes Cholesteatom der Schädelknochen, Deutsche Ztschr. f. Chir. **81**:90, 1906. Wertheimer, R.: Ueber ein Cholesteatom des Schädels, Fortschr. a. d. Geb. d. Röntgenstrahlen **38**:656 (Oct.) 1928.

15. Müller, J.: Ueber den feineren Bau und die Formen der krankhaften Geschwülste, Berlin, G. Reimer, 1838, p. 49. Mondschein, S.: Zur Kasuistik der Cholesteatom-tumoren der hinteren Schädelgrube, Wien. med. Wchnschr. **61**:576 (Feb. 25) 1911. Alpers, B. J., and Harrow, R.: Cholesteatoma Originating in the Skull Bones Causing Symptoms of Intracranial Pressure, Am. J. Surg. **18**:51 (Oct.) 1932. Rosenwasser, H.: Primary Cholesteatoma of the Occipital Bone, J. Mt. Sinai Hosp. **2**:56 (July-Aug.) 1935. King,[12e] case 2. Manasse, P.: Ueber chronische Mittelohreiterung und Cholesteatom (case 2), Deutsche med. Wchnschr. **38**:1172 (June 20) 1912.

16. Körner, O.: Ein Cholesteatoma verum in der hinteren Schädelgrube durch eine akute Mittelohreiterung infiziert und vereitert, Operation: Heilung, Ztschr. f. Ohrenh. **37**:352, 1900. Borchardt, M.: Cholesteatom der hinteren Schädelgrube, Arch. f. klin. Chir. **77**:892, 1905. Erdheim, J.: Ueber Schädelcholesteatome, Ztschr. f. Ohrenh. **49**:281, 1905.

17. Berger, W.: Ein atypisches Cholesteatom, Ztschr. f. Laryng., Rhin., Otol. **26**:39, 1935.

18. Giraldes, J.: Note sur les tumeurs dermoïdes du crâne, Gaz. méd. de Paris **37**:670, 1866.

19. Holsti, H.: Ett kolesteatom i hjärnan, Finska läk.-sällsk. handl. **28**:377; abstr., Jahresb. u. d. Leistung. d. ges. Med. **23**:263, 1888. Hofbauer: Cholesteatom des Stirnbeins, Deutsche med. Wchnschr. (Vereins Beilage) **29**:307 (Sept. 24) 1903.

20. Wrany, A., and Neureutter, T.: Casuistische Mittheilungen: IV. Cholesteatoma der Pia mater über dem Tuber corporis callosi bei einem fünf Jahre alten Mädchen, Oesterr. Jahrb. f. Pediat. **3**:22, 1872. Blasius, O.: Ein Fall von Epidermoid (Perlgeschwulst) der Balkengegend: Zugleich ein Beitrag zur Kenntnis der multiplen Hirnhernien, Virchows Arch. f. path. Anat. **165**:504, 1901.

21. (*a*) Parent-Duchatelet, A. J., and Martinet, L.: Recherches sur l'inflammation de l'arachnoïde cérébrale et spinale, Paris, Crevot, 1821, p. 439. (*b*) Cru-

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the suprasellar region,[22] the convexity,[23] the midline between the hemispheres [24] and the cerebellomedullary region,[25] it has always been

veilhier, J.: Anatomie pathologique du corps humaine, Paris, J.-B. Baillière, 1829, vol. 1, pt. 2, plate 6. (*c*) Wilks: Two Cases of Cholesteatomatous Tumor of the Brain, Tr. Path. Soc. London **10**:24, 1858-1859. (*d*) Bateman, F.: On Cholesteatoma, Arch. Med. **4**:316, 1867. (*e*) Eppinger, H.: Mittheilungen aus dem pathologisch-anatomischen Institut zu Prague, Vrtljschr. f. prakt. Heilk. **126**:33, 1875. (*f*) Price, J. A. P.: Cholesteatoma at the Base of the Brain, Tr. Path. Soc. London **38**:24, 1886-1887. (*g*) Glaeser, E.: Untersuchungen über Cholesteatome und ihre Ergebnisse für die Lehre von der Entstehung der Geschwülste, Virchows Arch. f. path. Anat. **122**:389, 1890. (*h*) Frank, C.: Ueber einem in der Duerener Irrenanstalt beobachteten Fall von Cholesteatom, Allg. Ztschr. f. Psychiat. **46**:30, 1890. (*i*) Haegi, H. J.: Ueber zwei Fälle von Cholesteatom der Hirnhäute, Zürich, J. Weiss, 1894. (*j*) Pfannmüller, W.: Ueber das meningeale Cholesteatom, Giessen, C. von Münchow, 1896. (*k*) Bailey, P.: Cruveilhier's "Tumeurs Perlées," Surg., Gynec. & Obst. **31**:390 (Oct.) 1920. (*l*) Kakeshita, T.: Zur Pathologie der Hirncholesteatome, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **27**:327, 1925. (*m*) Lohr, W., and Jacoby, W.: Mannsfaustgrosses, durch Operation geheiltes Cholesteatom der mittleren Schädelgrube, Zentralbl. f. Chir. **60**:1875 (Aug. 12) 1933. (*n*) Montgomery, G. L., and Finlayson, D. I. C.: Cholesteatoma of the Middle and Posterior Cranial Fossae, Brain **57**:177 (June) 1934. (*o*) Horrax, G.; Yorshis, M., and Lavine, G. R.: Calcified Intradural Cholesteatoma of Unusual Size in a Patient Showing Manic-Depressive Symptoms, Arch. Neurol. & Psychiat. **33**:1058 (May) 1935

22. Benda, C.: Zwei Fälle von Cholesteatom des Gehirns, Berl. klin. Wchnschr. **34**:167 (Feb. 22) 1897. Meyer, M.: Ueber Geschwülste der Hypophysengegend: Hypophysentumoren, Hypophysengangstumor, Hirnbasischolesteatom, Würzb. Abhandl. a. d. Gesamtgeb. d. Med. **22**:127, 1925. Olivecrona, H.: On Suprasellar Cholesteatomas, Brain **55**:122 (March) 1932.

23. Critchley and Ferguson,[12g] case 7.

24. (*a*) Petrina: Klinische Beiträge zur Localisation der Gehirntumoren, Vrtljschr. f. d. prakt. Heilk. **133**:95, 1877. (*b*) Critchley and Ferguson.[12g]

25. Le Prestre, F.: Observations sur des altérations organiques du cerveau: IV. Tumeur adipociriforme dévelopée dans le mésocephale, Arch. gén. de méd. **18**:19, 1828. Peyrot, M.: Tremblement mercuriel, sudamina. Tumeur comprimant le cervelet, ibid. **34**:620, 1834. Adams, W.: Cholesteatomatous Tumour of the Brain, Tr. Path. Soc. London **3**:44, 1850-1851. Peacock: Cholesteatomatous Tumour of the Brain, ibid. **5**:21, 1853-1854 (case of Dr. Thurnam). Nehrkorn, A.: Ein Fall von meningealer Perlgeschwulst, Beitr. z. path. Anat. u. z. allg. Path. **21**:73, 1897. Anschütz, W.: Ueber das Cholesteatom des Kleinhirnbrückenwinkels, Deutsche Ztschr. f. Chir. **201**:1, 1927. Kay, F. A., and Pack, G. T.: Cholesteatoma of Brain: Tumor of Unusual Size, Arch. Neurol. & Psychiat. **19**:446 (March) 1928. Kaufmann, E.: Lehrbuch der pathologischen Anatomie, translated by S. P. Reimann, Philadelphia, P. Blakiston's Son & Co., 1929, vol. 3, p. 1840. Virchow [13a] (case 3).

observed to arise from the meninges, as pointed out by Boström.[26d] The brain is involved only secondarily by pressure of the growth. The only exceptions to this statement are the cases in which the tumor has been noted in the lateral ventricles,[27] the third ventricle [28] or the fourth ventricle.[26] Here, the tissue of origin is apparently the choroid plexus. Of interest in connection with the brain is a case reported by DuVerney [29] in 1683. He quoted Bonn, who stated that Drelincourt had found a "steatoma" between the cerebrum and the cerebellum in the brain of a man dead of apoplexy. This is the earliest report of such a case that has come to my attention. Cholesteatoma has also been noted in the spinal cord.[30]

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26. Dumeril: Bull. Fac. de méd. de Paris **2**:32, 1807; quoted by Cruveilhier.[21b] (*b*) Spillman, P., and Schmitt, J.: Contribution à l'étude des tumeurs du quatrième ventricule, Arch. gén. de méd. **2**:182, 1882. (*c*) Toché, F.: Etude sur deux cas d'endothéliôme du cervelet, Thèse de Paris, no. 109, 1888. (*d*) Boström: Ueber piale Epidermoide, Dermoide, und Lipome und durale Dermoide, Zentralbl. f. allg. Path. u. path. Anat. **8**:1, 1897. (*e*) Knowles, B., and Peile, W. H.: A Case of Cholesteatoma of the Fourth Ventricle, Lancet **1**:1833 (June 28) 1902. (*f*) Ballance, C. A.: Some Points in the Surgery of the Brain and Its Meninges, New York, Macmillan & Co., 1908, p. 165, fig. 63. (*g*) Frick, K.: Ein Fall von Cholesteatom im IV Ventrikel, Frankfurt. Ztschr. f. Path. **6**:207, 1910-1911. (*h*) Shulgin, M. M.: Zwei Fälle von Cholesteatom in der Gegend des vierten Hirnventrikels, Sovrem. psikhiat. **5**:143, 1911; quoted by Meyer.[26i] (Five other cases cited from Russian literature.) (*i*) Meyer, W. R.: Zur Kasuistik der epidermoidalen Cholesteatome des Gehirns, Virchows Arch. f. path. Anat. **211**:161, 1913. (*j*) Bailey, P.: Further Observations on Pearly Tumors, Arch. Surg. **8**:524 (March) 1924. (*k*) Peter, R.: Cholesteatoma of Fourth Ventricle, Časop. lék. česk. **69**:1773 (Dec. 26) 1930.

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28. (*a*) Beneke: Zur Frage der meningealen Cholesteatome, Virchows Arch. f. path. Anat. **149**:95, 1897. (*b*) Thomas, J. J.: Cholesteatoma of the Brain, J. M. Research **6**:220, 1901. (*c*) Scholtz, F.: Einige Bemerkungen über das meningeale Cholesteatom im Anschluss an einen Fall von Cholesteatom des III Ventrikels, Virchows Arch. f. path. Anat. **184**:255, 1906. (*d*) Penfield, W.: Diencephalic Autonomic Epilepsy, Arch. Neurol. & Psychiat. **22**:358 (Aug.) 1929. (*e*) Loepp,[11e] (case 2).

29. DuVerney, J. G.: Traité de l'organe de l'ouie, Paris, E. Michallet, 1683, p. 189.

30. (*a*) Chiari: Centrales Cholesteatom des Dorsalmarkes mit Volkommen entwickelter auf- und absteigender Degeneration, Prag. med. Wchnschr. **8**:378, 1883. (*b*) Trachtenberg, M. A.: Ein Beitrag zur Lehre von den arachnoidealen Epidermoiden und Dermoiden des Hirns und Rückenmarks, Virchows Arch. f.

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Although there is general agreement that cholesteatoma of the tympanum can arise as a congenital tumor, there is nevertheless a rather large group of authors who feel that it is either predominantly or exclusively the result of infection.[31] Siebenmann [32] stated that he had never seen a primary cholesteatoma, and Politzer,[33] as an old man, stated that in all his life he had never seen such a tumor. Wittmaack,[34] whose work in otopathology is outstanding, stated that he had never seen the anlagen for such a tumor and that if one accepted these as the anatomic basis for the development of cholesteatoma, it must indeed be rare. Grunert,[35] on the same basis, reasoned that children should show the early stages of the tumor, but stated that in a large number of mastoidectomies in children under 1 year of age he had never encountered any evidence of it. Neither Heckner,[36] in 647 cases of chronic otorrhea,

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path. Anat. **154**:274, 1898. (*c*) Ivanoff, N. J.: Cholesteatom des Rückenmarks, J. nevropat. i psikhiat. (supp.) **3**:280, 1903. (*d*) Borst, M.: Geschwülste des Rückenmarks, Ergebn. d. allg. Path. u. path. Anat. **9**:514, 1904. (*e*) Lauterburg.[11g] (*f*) Marinesco, G., and Draganesco: Kyste epidermoide cholestéatomateux de la moelle épinière coexistent avec un processus syringomyélique: Contribution à l'étude de la syringomyélie, Rev. neurol. **31**:338 (Oct.) 1924. (*g*) Critchley and Ferguson,[24b] case 9.

31. Rokitansky, C.: Lehrbuch der pathologischen Anatomie, Vienna, Wilhelm Braumüller, 1855, vol. 1, p. 220. von Tröltsch, A.: Anatomische Beiträge zur Lehre von der Ohren-eiterung, Arch. f. Ohrenh. **4**:97, 1869; Lehrbuch der Ohrenheilkunde, Leipzig, F. C. W. Vogel, 1873, p. 422. Wendt, H.: Desquamative Entzündung des Mittleohrs, Arch. d. Heilk. **14**:428 and 551, 1873. Habermann, J.: Zur Entstehung des Cholesteatoms des Mittelohrs, Arch. f. Ohrenh. **27**:42, 1889. Bezold, F.: Cholesteatom, Perforation der Membrana Flaccida Shrapnelli, und Tubenverschluss, Ztschr. f. Ohrenh. **20**:5, 1890. Schmiegelow, E.: On the Perforations of the Membrana Flaccida Shrapnelli, with Remarks on the Formation of Cholesteatoma, Arch. Otol. **20**:228, 1891. Leutert, E.: Pathologisch-histologischer Beitrag zur Cholesteatomfrage, Arch. f. Ohrenh. **39**:233, 1895. Wingrave, V. H. W.: Notes on the Pathogeny of Cholesteatoma, Proc. Roy. Soc. Med. (Sect. Otol.) **3**:63 (June) 1910. Hellman, K.: Studien über das sekundäre Cholesteatom des Felsenbeins, Ztschr. f. Hals-, Nasen- u. Ohrenh. **11**:406, 1925. Lemaitre, F., and Aubin, A.: Cholestéatome de la fosse cérebélleuse, Arch. internat. de laryng. **6**:218, 1927. Tweedie, A. R., in discussion on McKenzie.[40f] Dundas-Grant, J., in discussion on McKenzie.[40f]

32. Siebenmann, F., quoted by Goerke, M.: Die Geschwülste des Ohres, in Denker, A., and Kahler, O.: Handbuch der Hals- Nasen- Ohrenheilkunde, Berlin, Julius Springer, 1926, vol. 7, p. 677.

33. Politzer, A., in discussion on Barujas: Arch. f. Ohrenh. **60**:101, 1904.

34. Wittmaack, K.: Ueber die normale und pathologische Pneumatization des Schläfenbeins, Jena, Gustav Fischer, 1918.

35. Grunert, K.: Ueber die Ergebnisse in der allgemein pathologischen und pathologisch-anatomischen Forschung des kranken Mittelohrs im letzten Jahrzehnt, Arch. f. Ohrenh. **60**:214, 1904.

36. Heckner, M.: Das Cholesteatom des Mittelohrs im Kindesalter, Munich, Kastner & Callwey, 1915, p. 18.

nor Ulrich,[37] in 458 cases of cholesteatoma, found any growth that could be designated as primary. Among 100,000 patients examined in Munich, Basle and Zurich Nager[38] found not a single instance of primary tumor. Lange[39] could find no such process in 100 temporal bones examined microscopically.

McKenzie,[40f] on the other hand, expressed the belief that all cholesteatomas are primary and that infection is a secondary occurrence.

In support of the theory that cholesteatoma may be congenital is cited a group of 5 cases,[41] in each of which a cholesteatoma was present in a malformed ear, with complete atresia of the canal. In 3 of the cases it was stated that the atresia was osseous. Obviously no possibility existed for an ingrowth of epidermis through a perforation of the tympanum. In each case the process was quiescent for a long time, in 1 instance for twenty-nine years, and attention was called to the ear only by secondary infection, which manifested itself by pain, swelling of the mastoid or fistula. The infection may have reached the tympanum via the eustachian tube or the blood stream. In 1 instance it was stated that the tube could not be sounded.

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37. Ulrich, K.: Zwei Beiträge zur Genese des Mittelohrcholesteatoms, Ztschr. f. Ohrenh. **75**:159, 1917.

38. Nager, F. R.: The Cholesteatoma of the Middle Ear: Its Etiology, Pathogenesis, Diagnosis and Therapy, Ann. Otol., Rhin. & Laryng. **34**:1249, 1925.

39. Lange, W.: Ueber die Entstehung der Mittelohrcholesteatome, Ztschr. f. Hals-, Nasen- u. Ohrenh. **11**:250, 1925.

40. (*a*) Hinton, J.: Sebaceous Tumor Within the Tympanum Originating on the External Surface of the Membrana Tympani, Bands of Membrane Occupying the Cavity, Guy's Hosp. Rep. **9**:264, 1863. (*b*) Urbantschitsch, V.: Ueber eine eigenthümliche Form von Epithelialauflagerung am Trommelfell und im ausseren Gehörgang, Arch. f. Ohrenh. **10**:7, 1876. (*c*) Küpper: Sectionsbefunde: IV. Cholesteatom des Trommelfelles (zufälliger Leichenbefund), ibid. **11**:18, 1876. (*d*) Wendt, H.: Ueber ein endotheliales Cholesteatom des Trommelfelles nebst Bemerkungen zur Histologie der Eigenschicht, Arch. d. Heilk. **14**:551, 1873. (*e*) Gruber, J.: A Text-Book of the Diseases of the Ear, translated by E. Law and C. Jewell, New York, D. Appleton & Co., 1893, p. 333. (*f*) McKenzie, D.: The Pathogeny of Aural Cholesteatoma, Proc. Roy. Soc. Med. **24**:332, 1931.

41. (*a*) Lüders, C.: Drei Fälle von retroaurikuläre Abszesse vortäuschenden Anschwellungen über dem Warzenfortsatze, Ztschr. f. Ohrenh. **66**:210, 1912. (*b*) Gumperz, R.: Ein Fall von Mikrotie, angeborener Gehörgangsatresie mit Cholesteatoma verum, Ztschr. f. Hals-, Nasen- u. Ohrenh. **2**:387, 1922. (*c*) de la Bernardie, C. B.: Imperforation du conduit auditif externe, cholestéatome primitif, evidement petro-mastoidien spontane: considérations étiologiques, Ann. d'oto-laryng., 1931, p. 952. (*d*) Goufas, G.: Malformation congénitale probable des oreilles externe et moyenne gauches, avec ostéite chronique du tympanal, réchauffée et extérioriséé, et coexistence cholestéatomateuse primitive, enkystée, de la cavité tympanale, ibid., 1932, p. 1362. (*e*) David, I.: Cholestéatome primitif du temporal avec malformation congénitale du pavillon de l'oreille et absence du conduit auditif externe, Rev. de laryng. **55**:511 (April) 1934.

A second series of reports [40] bearing on the same question concerns primary cholesteatoma of the drum membrane. In each of the cases one or more small white nodules were noted within the substance of the membrane, projecting into either the tympanum or the external canal. On removal and examination they were found to be made up of lamellated desquamated epithelial cells.

A third series [42] has to do with dermoids, or hairy cholesteatomas. These differ from cholesteatomas only in the inclusion of hair, glands or other tissue. In Pavlica's case tissue similar to glioma was present. They are simliar to cholesteatoma in that they originate from nests of misplaced ectodermal cells.

A fourth and final series [43] included 3 cases of cholesteatoma of the petrous apex in which the tympanum and the mastoid were not involved. In 1 case (Grahe) there was a war wound in which epidermis might have been carried in by a bullet, but in the other 2 the only explanation possible is a heterotopic displacement in early fetal life.

From the foregoing cases, in which the condition seems to controvert any doubt as to its being congenital, attention is now turned to a group of cases in which the evidence is not so complete, but in which, nevertheless, the history and physical findings point strongly toward a primary origin.

A great deal of effort has been expended in an attempt to define primary tumors belonging to this group, unfortunately without general agreement. Körner,[44] in 1899, laid down eight postulates by which primary cholesteatoma could be differentiated from "pseudo-cholesteatom," but my impression is that some of them would include too much.

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42. (*a*) Toynbee: Hairs in the Mastoid Cells, Tr. Path. Soc. London **17**:274, 1866. (*b*) Hinton, J.: Sebaceous Tumour and Hairs in the Tympanum of a Boy, ibid. **17**:275, 1866. (*c*) Wagenhäuser: Beiträge zur Pathologie und pathologischen Anatomie des Ohres, Arch. f. Ohrenh. **26**:1, 1888. (*d*) Scheibe, A.: Zwei Fälle von behaarter Granulationsgeschwulst im Mittelohr, Ztschr. f. Ohrenh. **25**:108, 1894. (*e*) Ribbert, M. W. H.: Geschwulstlehre für Aerzte und Studierende, Bonn, F. Cohen, 1904, p. 368. (*f*) Urbantschitsch, E.: Ueber einen Fall von scheinbar behaartem Mittelohrpolyp mit zentralem Cholesteatom bei vollständig eiterlosem Verlauf, Monatschr. f. Ohrenh. **43**: 291, 1909. (*g*) Grünwald, L.: Beiträge zur Kenntnis kongenitaler Geschwülste und Missbildungen an Ohr und Nase, Ztschr. f. Ohrenh. **60**:270, 1910. (*h*) Pavlica: Teratome congénital de l'os temporal, Bratisl. lekár. listy **9**:417, 1929.

43. Lehman: Ein ausserordentlich grosses Cholesteatom das die ganze Felsenbeinspitze zerstört hatte, Arch. f. Ohrenh. **91**:81, 1913. Grahe, K.: Abnorme Cholesteatombildung am Felsenbein, Ztschr. f. Laryng., Rhin., Otol. **20**:133, 1931. Beck, K.: Ueber einen operierten Fall von primären Cholesteatom an der Felsenbeinspitze, Ztschr. f. Hals-, Nasen- u. Ohrenh. **37**:117, 1934.

44. Körner, O.: Die eitrigen Erkrankungen des Schläfenbeins, in Die Ohrenheilkunde der Gegenwart und ihre Grenzgebiete, Wiesbaden, J. F. Bergmann, 1899, p. 119.

Kopetzky,[45] on the other hand, expressed the opinion that there is no method of distinguishing the true from the false type. Ruttin,[46] Linck,[47] Grünwald [42g] and Kahler [11d] expressed the belief that the presence of elastic fibers in the surrounding membrane indicates a primary growth, but Ulrich [37] and Hesse [48] rejected this view, since these fibers could be demonstrated in normal tympanic mucosa as well as in definitely secondary tumors. Since cholesteatoma takes years to develop, no symptoms other than deafness or tinnitus may appear until secondary infection prompts surgical intervention. For this reason, the otologic history and examination appear to offer the best method of determining whether or not a tumor is primary or secondary. Obviously this method has serious shortcomings, as Manasse,[49] Cushing [12e] and Lüders [41a] have pointed out, since infection may occur at any time and greatly reduce the number of cases in which the tumor is recognizable.

However, of the multitude of reports of cholesteatoma in the literature, 20 have been selected in which the growth was probably primary.[50]

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45. Kopetzky, S. J.: Cholesteatoma, Laryngoscope **43**:118 (Feb.) 1933.

46. Ruttin, E.: Zur Pathologie des Cholesteatoms, Ztschr. f. Hals-, Nasen- u. Ohrenh. **10**:59, 1924.

47. Linck, A.: Das Cholesteatom des Schläfenbeins, in Körner, O.: Die Ohrenheilkunde der Gegenwart und ihre Grenzgebiete, Munich, J. F. Bergmann, 1914.

48. Hesse, W.: Zur Differentialdiagnostik wahrer und falscher Ohrcholesteatome nebst kurzer Kritik der bisher beschriebenen kongenitalen Schläfenbeincholesteatome, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **114**:242 (Feb.) 1926.

49. Manasse, P.: Das Cholesteatom des Mittelohrs, bzw. des Felsenbeines, in Handbuch der pathologischen Anatomie des menschlichen Ohres, Wiesbaden, J. F. Bergmann, 1917, p. 64.

50. (*a*) Lucae, A.: Ueber Schwerhörigkeit bei grauer Degeneration des Rückenmarks, Arch. f. Ohrenh. **2**:305, 1867; (*b*) Beiträge zur Kenntnis der Perlgeschwulst des Felsenbeins, ibid. **7**:255, 1873. (*c*) Kuhn, A.: Das Cholesteatom des Ohres, Ztschr. f. Ohrenh. **21**:231, 1891. (*d*) Hansen: Ein Fall von Cholesteatoma verum des Ohres, München. med. Wchnschr. **55**:624 (March 24) 1908. (*e*) Hennebert: Echtes Temporalcholesteatom, Internat. Zentralbl. f. Ohrenh. **10**:15, 1912. (*f*) Lüders [41a] (case 2). (*g*) Urbantschitsch, E.: Cholesteatoma verum (?) des Mittelohrs mit Durchbruch in den Sinus: Thrombose des Sinus sigmoideus, petrosus superior, cavernosus und der Vena jugularis interna, Monatschr. f. Ohrenh. **46**:44, 1912. (*h*) Manasse [15f] (case 1). (*i*) Möller, J.: A Case of Primary Cholesteatoma Complicated by an Acute Otitis, Acta oto-laryng. **1**:309, 1918-1919. (*j*) Karlefors, J.: A Contribution to the Question of the Genesis of the Cholesteatoma in the Temporal Bone and Its Operative Treatment, ibid. **8**:417, 1925. (*k*) Trible, G. B., and Tibbets, A. P.: Cholesteatoma: Case Reports, Laryngoscope **36**:512 (July) 1926. (*l*) Rateau, J., and Rouquette: Le cholestéatome primitif total du Rocher, Rev. de laryng. **48**:353 (May) 1927. (*m*) Karlefors, J.: Cholesteatom mit unverletztem Trommelfell: Radicaloperation ohne Plastik: Heilung, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **12**:105, 1928.

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In none of these cases was there a history of otorrhea for more than five months prior to operation, and in the majority, the discharge had been present for only two or three weeks. In some of the cases the drum membrane was imperforate. In addition, Körner [44] reported 5 cases and Linck,[47] 2 cases in which a diagnosis of primary cholesteatoma had been made, but sufficient data were not recorded; so the cases could not be included in the aforementioned group.

In regard to the etiology of the condition, the majority of authors have agreed with the theory of Remak,[51] who believed that dermoid and related tumors arise from dislocated follicles of skin during early embryonic life. Küster [52] and Mikulicz [1b] stated that the anomalous planting of ectodermal cells may be similar to that in branchiogenic cysts, the latter suggesting, in addition, an origin from the primordial otic vesicle. Böttcher [53] suggested an origin from the aquaeductus vestibuli. Virchow [13a] and his student Prahl [54] expressed the belief that the epithelial elements arose "out of and in, connective tissue," and specifically, in the brain, from endothelium of the pia or a plexus. Paget [55] expressed the belief that the condition is partly hereditary.

While I believe that primary cholesteatoma of the ear arises from ectodermal cells, I do not believe that these are anomalously placed; I think rather that they are normally placed cells which have taken on an anomalous function. To indicate the method of their arrival in the

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(*n*) Grove, W. E.: Primary Cholesteatoma of the Temporal Bone, Arch. Otolaryng. **10**:398 (Oct.) 1929. (*o*) Tanturri, V.: Su di un caso di colesteatoma gigante dell'orrechio: Considerazioni isto-patologiche, Rassegna ital. di oto-rino-laring. **3**:257 (Sept.-Oct.) 1929. (*p*) Carrari, G. G.: Di un caso di colesteatoma primitivo dell'orrechio: Tromboflebite necrotica. Setticopiemia. Radicale alla Barany. Guarigione, Valsalva **6**:587 (Sept.) 1930. (*q*) Scott, S., in discussion on McKenzie.[40f] (*r*) Just, T. H., in discussion on McKenzie.[40f] (*s*) Lindsay, J. R.: Cholesteatoma Associated with an Isolated Perforation in Shrapnell's Membrane (case 2), Arch. Otolaryng. **20**:47 (July) 1934. (*t*) de Mare, G.: Le cholestéatome "congénitale" du temporal, à propos d'un fait clinique, Acta oto-laryng. **22**:163, 1935.

51. Remak, R.: Ein Beitrag zur Entwicklungsgeschichte der krebshaften Geschwülste, Deutsche Klin. **6**:170 (April 22) 1854.

52. Küster, E., in discussion on Virchow, R.: Perlgeschwülste des Felsenbeins, Berl. klin. Wchnschr. **26**:217 (March 11) 1889.

53. Böttcher, A.: Ueber den Aqueductus vestibuli bei Katzen und Menschen, Arch. f. Anat., Physiol. u. wissensch. Med., 1869, p. 372.

54. Prahl, P.: Ueber die Perlgeschwülste mit besonderer Berücksichtigung ihres Vorkommens im Felsenbein, Berlin, G. Lange, 1867.

55. Paget, J.: Lectures on Surgical Pathology, London, Longman, Brown, Green & Longmans, 1853, p. 85.

tympanum, it will be necessary to review the phylogeny and ontogeny of the ear somewhat in detail.

Following the description of epibranchial organs in fish by Leydig [56] in 1868, Van Wijhe [57] and Beard [58] studied their development and found that they were formed by a union of the cranial ganglions with ectodermal thickenings or placodes. Greil,[59] studying Cerotodus, described the placodes as occurring at the point of union of the pharyngeal pouches with the branchial clefts, but Landacre,[60] using embryos of Lepidosteus, found that while the epibranchial placode formed in close proximity to this union, it could nevertheless be differentiated from it. Landacre also pointed out, as did Froriep,[61] that there are two placodes with which the facial ganglion makes contact. The dorsal placode becomes part of the lateral line system, while the ventral placode becomes detached from the ectoderm and becomes incorporated in the facial ganglion. This double origin of the facial ganglion had also been noted by Greil [59] and by von Kupffer.[62] In some forms, according to Agar [63] and Vitali,[64] the cellular mass resulting from the union of the placode with the facial ganglion gives rise to an epithelial vesicle called the spiracular organ. This structure in an adult lies near the quadrate [64a] and the squama and is supplied by a branch of the facial nerve. It contains a sensory epithelium in which the nerve fibers terminate.

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56. Leydig, F.: Ueber Organes eines sechsten Sinnes, Dresden, E. Blochmann & Sohn, 1868.

57. Van Wijhe, J. W.: Ueber die Mesodermsegmente und die Entwicklung der Nerven des Selachierkopfes, Versl. d. k. Akad. v. Wetensch. **18**:71, 1883.

58. Beard, J.: The System of Branchial Sense Organs and Their Associated Ganglia in Ichthyopsida: A Contribution to the Ancestral History of Vertebrates, Quart. J. Micr. Sc. **26**:95 (Nov.) 1885.

59. Greil: Ueber die Entstehung der Kiemendarmderivate von Cerotodus, Verhandl. d. anat. Gesellsch. **20**:115, 1906.

60. Landacre, F. L.: The Epibranchial Placodes of Lepidosteus Osseus and Their Relation to the Cerebral Ganglia, J. Comp. Neurol. **22**:1 (Feb.) 1912.

61. Froriep, A.: Zur Entwicklungsgeschichte der Kopfnerven, Verhandl. d. anat. Gesellsch. **5**:55, 1891.

62. von Kupffer, C.: Die Entwickelung der Kopfnerven der Vertebraten, Verhandl. d. anat. Gesellsch. **5**:22, 1891.

63. Agar, W. E.: The Spiracular Gill Cleft in Lepidosiren and Protopterus, Anat. Anz. **28**:298 (March 12) 1906.

64. Vitali, G.: Il comportamento dello organo della prima fessura branchiale (placode epibranchiale) nei Selaci, Monitore zool. ital. **36**:122, 1925.

64a. The quadrate is the osseous or cartilaginous structure on both sides of the head in fish and birds; it articulates with the lower jaw.

A similar state of affairs exists in amphibia.[65] The facial ganglion is made up by a union of the ectodermal placode with a portion of the ganglionic ridge, although no structure comparable to the spiracular organ is found.

In birds the process is carried a bit further. Kastschenko[66] and others[67] have described the union of the facial ganglion with the first epibranchial placode in the chick, thus carrying on the analogy with lower forms. Kastschenko noted that the result of this union is an epithelial vesicle, which is in contact with the first pharyngeal pouch and soon opens into it. A gradual separation then takes place, so that by the eighth day of incubation the vesicle is again separate from the primordial tympanum but attached to it by a thin pedicle with a scarcely recognizable lumen. The epithelium of the vesicle is thickened on the side which is joined by a branch from the geniculate ganglion.

Vitali took up the study at this point and showed that in the cock sparrow[68] and in other birds[69] this vesicle persists into adult life as a definite sensory organ which is analogous to the spiracular organ of fish.[64] It is absent, however, in certain nocturnal birds.[70] In adult animals the vesicle is oval and is situated near the articulation of the quadrate and the lateral occipital bone. It lies just above the superior wall of the tympanum, from which it is separated by a small amount

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65. Götte, A.: Entwicklungsgeschichte der Unke, Leipzig, 1875. Johnson, A., and Sheldon, L.: On the Development of the Cranial Nerves of the Newt, Proc. Roy. Soc. London **40**:94 (Jan. 28) 1886. Houssay, M.: Sur la métamérie de la tête chez l'axolotl, Compt. rend. Soc. de biol. **1**:691 (Nov. 30) 1889. Drüner, L.: Studien zur Anatomie der Zungenbein- Kiemenbogen- und Kehlkopfmusculatur der Urodelen, Zool. Jahrb. (Abt. f. Anat.) **19**:361, 1904. Brachet, A.: Recherches sur l'ontogénèse de la tête chez les amphibiens, Arch. de biol., Paris **23**:165, 1908. Marcus, H.: Beiträge zur Kenntnis der Gymnophionen: III. Zur Entwicklungsgeschichte des Kopfes, Morphol. Jahrb. **4**:105, 1910. Smith, L. W., quoted by Kingsbury.[84c]

66. Kastschenko, N.: Das Schlundspaltengebiet des Hühnchens, Arch. f. Anat. u. Entwcklngsgesch., 1887, p. 258.

67. Mall, F.: Development of the Eustachian Tube, Middle Ear, Tympanic Membrane and Meatus of the Chick, in Studies from the Biological Laboratory, Johns Hopkins University, Baltimore, Johns Hopkins Press, 1888, vol. 4, p. 185; The First Branchial Cleft of the Chick, Johns Hopkins Univ. Circ. **7**:38 (Feb.) 1888. Golowine, E.: Sur le dévelopment du système ganglionnaire chez le poulet, Anat. Anz. **5**:119 (Feb. 15) 1890. Goronowitsch, N.: Untersuchungen über die Entwicklung der sog. "Ganglienleisten" im Kopfe der Vögelembryonen, Morphol. Jahrb. **20**:187 (Aug. 4) 1893.

68. Vitali, G.: Di un interessante derivato della prima fessura branchiale nel passero (nota preliminare), Anat. Anz. **39**:219 (June 21) 1911.

69. Vitali, G.: Di un interessante derivato dell'ectoderma della prima fessura branchiale nel passero: Un organo nervoso di senso nell'orecchio medio degli uccelli, Anat. Anz. **40**:631 (March 8) 1912.

70. Vitali, G.: Sul comportamento dell'organo della prima fessura branchiale in "Athene Noctua," Ricerche di morphol. **3**:153, 1923.

of connective tissue. Its cavity, containing a fluid giving the reaction of mucin, does not communicate with that of the tympanum. Vitali called it the "paratympanic organ." Its wall is made up of a single layer of cells, except in the sensorium,[71] where there are two or more layers. Sensory and supporting elements can be made out, the former having sensory hairs. A branch of the facial nerve enters the sensorium, where branched and unbranched fibers pass to the sensory cells. These fibers do not originate in the geniculate ganglion, according to Vitali [72] and Federici,[73] but arise in the vestibular nuclei and are merely carried to their destination by the facial nerve.

Vitali [74] expressed the belief that the paratympanic organ has to do with flight, since its destruction is followed by lack of tonus in the wings and by abnormalities in flying. Benjamins [75] repeated the work with negative results.

In mammals an epibranchial organ was first described in 1885 by Froriep,[76] although Urbantschitsch [77] had figured the placode in 1877, without apparently appreciating its significance. Mall [78] described it in dog embryos. Froriep [76] noted in 8.8 mm. calf embryos a spindle-shaped ectodermal thickening, or placode, at the dorsal end of the first branchial cleft, which is in contact with the facial ganglion. A similar thickening is present over the second cleft, which is in contact with the ganglion petrosum of the glossopharyngeal nerve. At 12 mm. the placode has

71. Vitali, G.: Di un nuovo organo nervoso di senso nell'orecchio medio degli uccelli: Ulteriore destino dell'organo della prima fessura branchiale, Internat. Monatschr. f. Anat. u. Physiol. **30**:363, 1914.

72. Vitali, G.: Sui disturbi funzionali e sulle lesioni istologiche dipendenti dalla distruzione dell'organo nervoso di senso da me descritto nell'orecchio medio degli uccelli, Atti d. r. Accad. d. fisiocrit. in Siena **7**:291 (Dec. 4) 1915.

73. Federici, F.: Ueber die peripherische Ausbreitung des VIII Schädelnervenpaares bei den Vögeln und über die Bedeutung der Lagena, Anat. Anz. **61**:449 (Oct. 25) 1926; Ueber die Innervation des von Vitali entdeckten Sinnesorgans im Mittelohr der Vögel (sogen. paratympanisches Organ), ibid. **62**:241 (Jan. 26) 1927.

74. Vitali, G.: La façon de se comporter du placode de la prémière fente branchiale (placode epibranchiale) dans la série des vertébrés, Arch. ital. de biol. **76**:94 (March 30) 1926.

75. Benjamins, C. E.: Recherches sur l'organe paratympanique de Vitali chez les oiseaux, Rev. de laryng. **47**:58 (Jan. 31) 1926.

76. Froriep, A.: Ueber Anlagen von Sinnesorganen am Facialis, Glossopharyngeus und Vagus, über die genetische Stellung des Vagus zum Hypoglossus, und über die Herkunft der Zungenmusculatur: Beitrag zur Entwickelungsgeschichte des Säugethierkopfes, Arch. f. Anat. u. Entwcklngsgesch., 1885, p. 1.

77. Urbantschitsch, V.: Ueber die erste Anlage des Mittelohres und des Trommelfelles, Mitth. a. d. embryol. Inst. d. k. k. Univ. in Wien **1**:1, 1877.

78. Mall, F. P.: The Branchial Region of the Dog, Johns Hopkins Univ. Circ. **7**:39 (Feb.) 1888; The Branchial Clefts of the Dog, with Special Reference to the Origin of the Thymus Gland, in Studies from the Biological Laboratory, Johns Hopkins University, Baltimore, John Hopkins Press, 1888, vol. 4, p. 193.

become a funnel-shaped invagination, which is in contact both with the facial ganglion and with the dorsal prolongation of the first pharyngeal pouch. At 15 mm. the invagination is a little wider and flatter but is still in contact with the first pharyngeal pouch.

Rabl [79] carried the work further from this point and showed that in the guinea-pig the invagination becomes deeper and finally a small vesicle is formed. It then retracts from the epidermis, with which it remains in connection by a short solid cord of cells. In the meantime it has established an opening into the primordial tympanum. Figure 1, which was taken from the article by Rabl, shows three serial sections, indicating the method by which this communication is accomplished. A little later the mass of epithelial cells fuses with the dorsal diverticulum of the first pharyngeal pouch (primordial tympanum), where it remains for some time as a thickened epithelium.

Rabl also studied the mole, in which a similar condition appears.

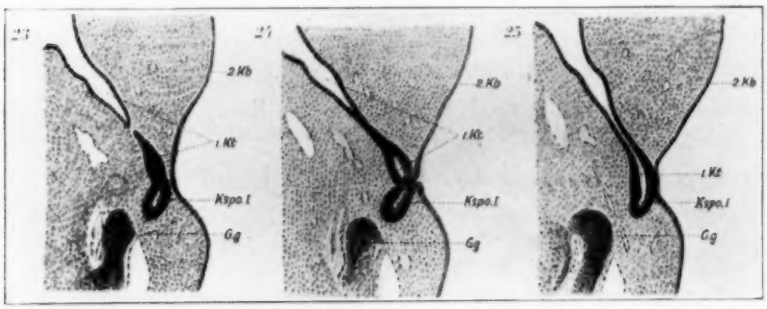

Fig. 1.—From Rabl.[79] Three serial sections, showing communication between the first pharyngeal pouch and the first epibranchial organ in the guinea-pig. *2 Kb* indicates the second branchial arch, *1 Kt* the first pharyngeal pouch, *Kspo'* the first epibranchial organ and *Gg* the geniculate ganglion.

A true paratympanic organ similar to that in birds does not exist in mammals, with the single exception of Vesperugo pipistrellus, a chiropter.[80] In view of Vitali's [74] theory that the organ has to do with flight, it is interesting to note that the only mammal possessing it is likewise a member of the only group of mammals which fly.

With the foregoing considerations in mind, it would be expected that the human embryo would present a similar set of circumstances. Chiarugi [81] in 1889 noted that in a human fetus at the beginning of

79. Rabl, H.: Die Entwicklung der Derivate des Kiemendarmes beim Meerschweinchen, Arch. f. mikr. Anat. **82**:79 (May 30) 1913.

80. Vitali, G.: Il comportamento dell'organo della prima fessura branchiale negli anfibi, nei rettili e nei mammiferi, Ricerche di morphol. **4**:191, 1924.

81. Chiarugi, G.: Anatomie d'un embryon humain de la longeur de mm. 2.6 en ligne droite, Arch. ital. de biol. **12**:273, 1889.

the fourth week the facial ganglion made contact with the thickened, slightly invaginated ectoderm of the dorsal segment of the first branchial cleft. Giglio-Tos,[82] in an embryo of about 17 days, noted a similar condition. Figure 2, which is reproduced from his article, shows the pharyngeal pouch on the left of the fetus in contact with a mass of cells derived from the ectoderm *ge* and the acousticofacial ganglion *af*. The dorsolateral placode *pf* corresponds to the anlage of the lateral line organs in fish. On the right side of the human fetus, in a position which is a little more posterior than that assumed by these structures in the fish, the pharyngeal pouch is in contact with the invaginated placode.

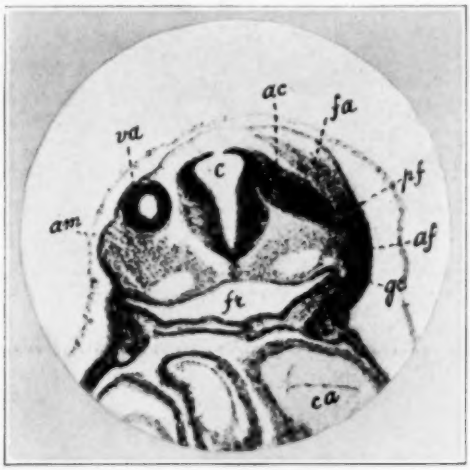

Fig. 2.—From Giglio-Tos.[82] An explanation appears in the text.

Hammar [83] and others [84] have also described the epibranchial organ in man. Janosik [85] was unable to find any union of the facial ganglion

82. Giglio-Tos, E.: Sui primordi dello svilluppo del nervo acustico-faciale nell' Uomo, Anat. Anz. **21**:209 (May 14) 1902.

83. Hammar, J. A.: Studien über die Entwicklung des Vorderdarms und einiger angrenzenden Organe: I. Allgemeine Morphologie der Schlundspalten beim Menschen. Entwicklung des Mittelohrraumes und des äusseren Gehörganges, Arch. f. mikr. Anat. u. Entwcklngsgesch. **59**:471, 1902.

84. (*a*) Streeter, G. L.: Nerves of Special Sense Organs (Special Somatic Sensory), in Keibel, F., and Mall, F. P.: Manual of Human Embryology, Philadelphia, J. B. Lippincott Company, 1912, vol. 2, p. 129. (*b*) Keibel, F.: The Epibranchial Sense-Organs, ibid., p. 182. (*c*) Kinsbury, B. F.: The Development of the Human Pharynx: I. The Pharyngeal Derivatives, Am. J. Anat. **18**:329, 1915.

85. Janosik, J.: Zwei junge menschliche Embryonen, Arch. f. mikr. Anat. **30**: 559, 1887.

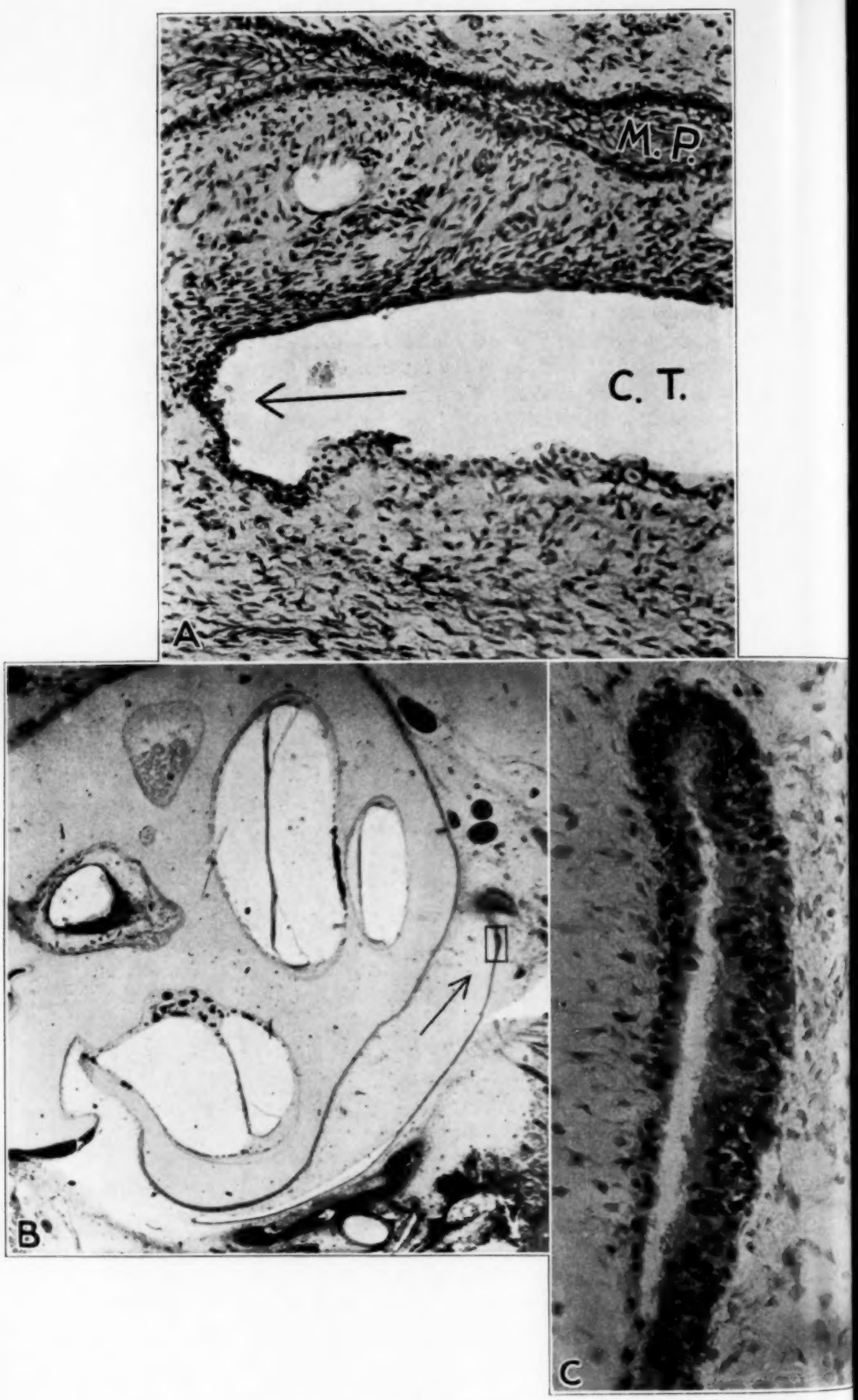

Fig. 3.—*A*, section of 4 month fetus, showing thickening of the mucosa in the posterior pole of the tympanum (*C T*). The arrow points to the thickened zone. *M P* indicates the meatal plate. *B*, vertical section from ear of twin of the fetus shown in *A*. *C*, high magnification of the portion indicated by the arrow in *B*.

and the ectoderm in two human fetuses of about 3 mm. (about 15 days), and Weigner [86] could find no connection in the 3 mm., 10 mm. and 13 mm. fetus. Since a vesicle has never been noted in man and the union of the facial nerve with the ectoderm disappears soon after the 8 mm. stage, one concludes that this is indeed an extremely ephemeral

Fig. 4.—Low power view of section of a 5½ month fetus, showing a small mass of epidermal cells in the upper lateral portion of the tympanum, just medial to the malleus. The arrow indicates the mass.

structure. While the later stages have as yet received little attention, it is probable that the placode remains for some time as an epithelial

86. Weigner, K.: Bemerkungen zur Entwicklung des Ganglion acoustico-faciale und des Ganglion semilunare, Anat. Anz. **19**:145 (March 23) 1901.

thickening in the dorsal pole of the tympanum. Figure 3 *A* is from a 4 month fetus which I observed and shows, in the dorsolateral position of the tympanum (indicated by the arrow), a definite thickening of the epithelium. The cells in this region appear similar to those of the basal layer of the meatal plate *mp* but quite unlike those of the remainder of the tympanic mucosa. This illustration is from a paraffin section cut

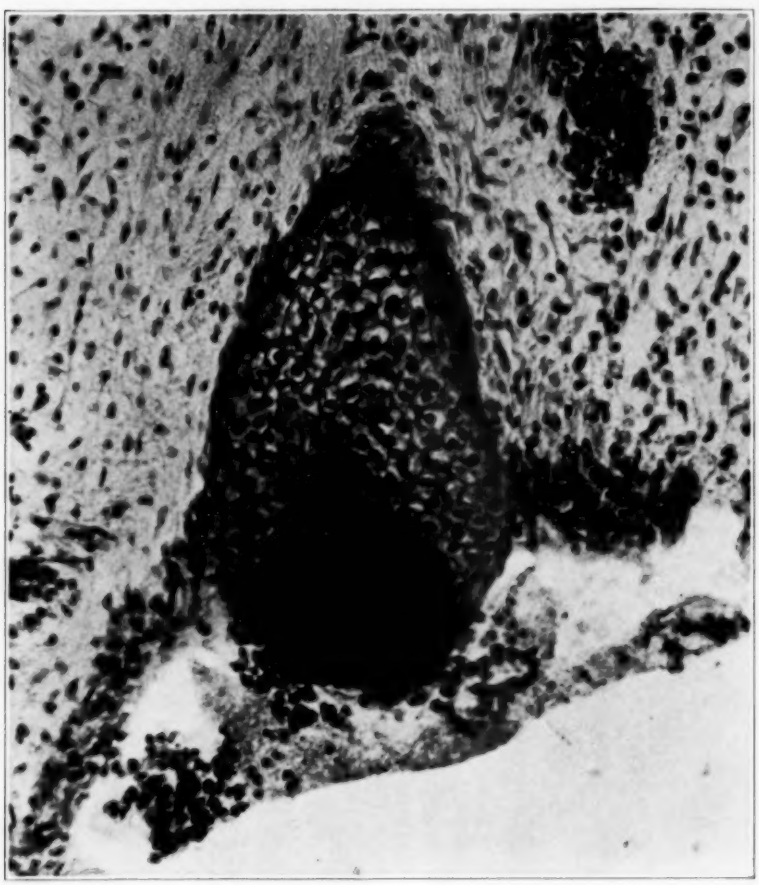

Fig. 5.—High power view of the small epidermal mass in the tympanum shown in figure 4.

5 microns thick, and it is improbable that the layering is due to artefact. The thickening is present in only thirty sections (150 microns). Figure 3 *B* and *C* are taken from the twin of the fetus shown in figure 3 *A* and are vertical sections. They show well the difference in thickness between the upper and the lower portion of the tympanic mucosa. Several other fetuses in my collection have exhibited the same type of

thickening in this region. Under normal conditions the cells gradually become transformed into a single layer of flat cells similar to the entodermal lining of the tympanum, so that by the time of birth no differentiation between the two types of cell can be made.

The question presenting itself at this point is: What should prevent these ectodermal cells from behaving occasionally as other ectodermal cells and forming epidermis? Figure 4 is from a personally observed 5½ month fetus, in which this was actually happening. In the dorsolateral pole of the tympanum, just medial to the neck of the malleus is a small splinter of epidermal cells, the distal portion showing cornification and desquamation. Figure 5 is a high power view of the focus, in the tympanum around which are leukocytes and histiocytes, apparently called out in response to the irritation of the epidermal scales.

The only other reported instance of a similar process of which I am aware is that of Přecechtěl,[87] who noted epidermal splinters in the attic, promontory, hypotympanic recess and cochlea of a new-born infant. There was also a malformation of the external ear. No pictures were given, but the surface of the cells was said to be keratinizing and desquamating. In this case there was, in addition to the normally placed epidermis in the attic, abnormally placed tissue in the other regions mentioned.

In the further growth of these epidermal foci, desquamation continues, and finally the middle ear and the mastoid become filled with squames and crystals of cholesterol. The process is slow, and no pain results. The hearing is also slowly reduced, and the patient may be entirely unaware of its loss until deafness is well marked. When infection occurs, either hematogenously or through the tube, pus may burrow through the drum, the mastoid cortex or the dura. Operation reveals the tympanum or mastoid or both filled with a necrotic mass. Often the entire mastoid is excavated, and in some instances even the wall of the labyrinth has been destroyed.

SUMMARY

In the foregoing discussion the wide distribution of primary cholesteatoma is noted, with special reference to its occurrence in the ear. To controvert those who feel that cholesteatoma of the tympanum must always be associated with infection, four groups of cases are cited in which little doubt of congenital origin can be entertained. The opinion is then expressed that the best method of distinguishing a primary from a secondary cholesteatoma is by anamnesis and examination, on the basis of which twenty additional cases are cited from the literature. A review of the relationship between the first pharyngeal pouch and

87. Přecechtěl, A.: A Contribution to the Development of Congenital Atresia of the Ear with Cholesteatoma, Acta oto-laryng. **14**:123, 1930.

the first epibranchial placode in fish, amphibia, birds and mammals is made, leading to a discussion of a similar relationship in man. From this, it is deduced that epidermal cells are normally present in the dorsolateral pole of the tympanum and that normally they become transformed into epithelial cells. Occasionally, however, they persist in their ectodermal quality and produce skin. The desquamation which results forms the cholesteatoma.

CONCLUSION

Primary cholesteatoma of the middle ear and the mastoid may result from the anomalous functioning of normally placed epidermal cells.

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LATE RESULTS FOLLOWING OPERATION FOR CARCINOMA OF THE LARYNX

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Late results of the operative treatment for carcinoma of the larynx are best learned if a group of consecutive cases are studied rather than any number of individual or selected cases. For this reason, the results of all the operations for carcinoma of the larynx performed between 1912 and 1932 at the Johns Hopkins Hospital were studied.

The symptom of gradually increasing painless hoarseness coming on in adult life is too well known to the laryngologist, too little heeded by the laity and at times too lightly considered even by members of the medical profession. Pain and dyspnea are associated with inoperable cancers. Hoarsneess is the danger signal, and until carcinoma can be definitely ruled out, it should be considered the cause of the hoarseness. In justice to the patient the worst conditions should be thought of first. Early diagnosis of carcinoma of the larynx is imperative, and before laryngotomy or laryngectomy is performed the diagnosis of carcinoma must be confirmed by microscopic examination of the tumor.

All too frequently the condition is inoperable. However, a condition which one surgeon may think operable another may think inoperable. If in the desire to keep his operative results favorable a surgeon is too cautious, he deserves blame. Some surgeons consider that no condition is ever inoperable, but the limit is well defined by others.

Hajek [1] at his clinic operates in all cases in which it seems technically possible. He has stated that in certain cases the best treatment is thyrotomy, but he does not hesitate to change to laryngectomy if the growth proves to be larger than expected. Hajek and Heindl [2] in a recent report on 393 patients with carcinoma of the larynx surgically treated stated that 44 per cent of the patients after laryngofissure or partial

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Read before the Section on Laryngology, Otology and Rhinology at the Eighty-Seventh Annual Session of the American Medical Association, Kansas City, Mo., May 14, 1936.

1. Hajek, M.: Wandlungen in der Indikation der chirurgischen Larynxoperationen beim Kehlkopfkrebs, Wien. med. Wchnschr. **80**:16-31, 1930.

2. Hajek, M., and Heindl, A.: Statistics in Three Hundred and Ninety-Three Cases of Cancer of the Larynx Surgically Treated, Monatschr. f. Ohrenh. **69**:385, 1935.

resection were well for longer than three years, with a postoperative mortality of 15 per cent. Thirty-five per cent of the 89 patients with internal cancer treated by laryngectomy were well for longer than three years. Eleven per cent of 152 patients with external cancer were well for over three years.

MacKenty [3] saw 143 patients with carcinoma of the larynx between 1917 and 1922. In 46 cases he considered that the condition was operable. He had much greater success with laryngectomy than with thyrotomy. Three of his patients with incipient carcinoma required laryngectomy with serial section of the larynx, showing that the growth could not have been removed by thyrotomy. In general the portion of the growth seen is one third of the actual size of the mass, two thirds being invisible. Later MacKenty stressed the danger signal progressive painless hoarseness and stated: "Radium has been an utter failure for treatment in cases of squamous cell laryngeal cancer (i.e., in 96 per cent of laryngeal cancers). Its false promise is luring thousands beyond the aid of surgical procedures." Thirty-five per cent of 230 patients treated surgically for incipient intrinsic cancer showed recurrence after thyrotomy; 3 per cent showed recurrence after laryngectomy. Twenty-five per cent of the patients with intrinsic moderately advanced carcinoma showed recurrence after laryngectomy. There was recurrence in 35 per cent of the borderline cases (nearly extrinsic) and in 100 per cent of the cases of extrinsic carcinoma. Laryngectomy was fatal in 3 per cent of cases.

Thomson [4] reported that 40 of his 55 patients were alive and free from recurrence three years after operation. Laryngofissure was used only when the vocal cord was movable and the growth was limited to one cord. Later Thomson reported on all his patients treated by laryngofissure or partial laryngectomy—70 with intrinsic carcinoma (63 men and 7 women). Thirty-four were still alive, 18 had died from other causes and 7 had died as a result of a malignant growth in other parts. There were 11 recurrences, 8 developing in the first year. In another review Thomson showed the results obtained in relation to the

3. MacKenty, J. E.: Operative Treatment of Cancer of the Larynx, J. Laryng. & Otol. **39**:67-78, 1924; Cancer of the Larynx, Arch. Otolaryng. **3**:205-232 (March); 305-337 (April) 1926; Laryngeal Cancer: Early Diagnosis and Treatment, ibid. **9**:237-244 (March) 1929.

4. Thomson, St. Clair: Cancer of the Larynx, Arch. Otolaryng. **3**:37-42 (Jan.) 1926; Intrinsic Cancer of the Larynx Operated on by Laryngofissure: Immediate and Ultimate Results, Brit. M. J. **1**:743-745, 1928; Arch. Otolaryng. **8**: 377-385 (Oct.) 1928; Intrinsic Cancer of the Larynx: Occurrence of Impaired Mobility of the Affected Cord and Its Value in Diagnosis and Prognosis, Lancet **2**:220-221, 1928; Intrinsic Cancer of the Larynx: Lasting Cure in Seventy-Six Per Cent of Cases by Laryngo-Fissure, Canad. M. A. J. **21**:4-8, 1929; The Diagnosis of Cancer of the Larynx, J. Laryng. & Otol. **46**:31-36, 1931.

motility of the cord after laryngofissure. Thirty-one (84 per cent) of the 37 patients with a mobile cord were free from recurrence for three years. Fifteen (75 per cent) of the 20 patients with impaired movement were free from recurrence for three years. Forty per cent of the 10 patients with fixation of the cord were free from recurrence after three years. A cord may be mobile and yet harbor carcinoma. Impaired motility or fixation renders the prognosis more unfavorable. Thomson gave a complete summary of the diagnosis of carcinoma of the larynx. Two of the 63 patients operated on without biopsy were found to have a noncancerous growth, one being tuberculous and the other keratotic. Tuberculosis of the larynx is mistaken for carcinoma more often than is any other lesion. Tuberculosis, carcinoma and syphilis may be present together.

Jackson [5] has stressed the danger of hoarseness and has emphasized the necessity of a complete view of the anterior commissure in making a diagnosis of carcinoma of the larynx. He reported 120 consecutive laryngofissure operations with no postoperative deaths. Of 127 patients, 102 were free from recurrence after one year, 2 were alive thirty-one and twenty-five years, respectively, after operation, 4 died of other diseases after twenty years, 3 died of carcinoma of the abdominal organs and 2 died of carcinoma of the tongue, with no laryngeal recurrence. In cases of early intrinsic carcinoma at least 80 per cent of relative cures can be obtained by the comparatively minor and nonmutilating operation of laryngofissure. Jackson described the cases which he considered suitable for laryngofissure and reported on 74 patients who lived for five or more years. Of those who could be traced, 32 per cent were alive and well for from five to thirty-one years; 5 died from recurrences and 2 from carcinoma of the other regions, without local recurrence. He again made a plea for early diagnosis and operation.

Tucker [6] reported on 30 patients treated by laryngofissure. Twenty-five were well after one or more years, 1 was well after seven years and 8 were well after four years. There were 2 recurrences. He reviewed the diagnosis and treatment in these cases and made a plea for early operation.

New and Waugh [7] reported on 32 patients treated by laryngofissure, with 82 per cent of cures. There were 73 patients treated by laryn-

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5. Jackson, C.: Cancer of the Larynx: Its Treatment by Laryngofissure, South. Surgeon **1**:223-229, 1932; Cancer of the Larynx: Its Curability by Laryngofissure, Surg., Gynec. & Obst. **58**:431-432, 1934.

6. Tucker, Gabriel: Early Intrinsic Cancer of the Larynx: Diagnosis and Treatment; Observations on Laryngofissure as Method in Series of Cases, Ann. Otol., Rhin. & Laryng. **41**:36-51, 1932; Cancer of the Larynx, Surg., Gynec. & Obst. **46**:303-1028.

7. New, G. B., and Waugh, J. M.: The Curability of Carcinoma of the Larynx, Surg., Gynec. & Obst. **58**:851-884, 1934.

gectomy, with 56 per cent of cures for five years. New and Figi [8] advised against any set operative procedure. The operation should be exploratory, so that all cancerous tissue is completely removed. With this method there were 82 per cent of five year cures after thyrotomy and 56 per cent of five year cures after laryngectomy with a two stage operation.

Colledge [9] reports on 14 laryngectomies. Seven of the patients were well from one to four years later.

Fraser and Watson [10] reported on 14 cases of laryngofissure with 7 recoveries. They remarked that excision of the larynx should have been carried out in several cases instead of laryngofissure.

Finzi and Harmer [11] reported on 15 cases (in 13 the diagnosis was confirmed by biopsy) in which treatment was given with radium needles after window resection of the thyroid cartilage. The patients were compelled to wear a tracheotomy tube for six weeks because of edema of the larynx. There were 14 men and 1 woman. In 6 of the 8 cases of intrinsic carcinoma the growth disappeared in from one to three years. In 2 cases in which glandular involvement rendered the condition inoperable there was 1 recurrence after disappearance of the growth. One mass shrank considerably, but the patient died in fifteen months.

Graham,[12] on the other hand, stated: "Window resection of the cartilage for the sake of treatment of carcinoma of the larynx by x-ray and radium is more of a menace to the life of the patient than a help and should be discouraged."

Pancoast [13] in 1924 stated: "We have never cured a proven case by radiation alone. Many disappear only to recur hopelessly. We freely acknowledge the superiority of improved surgical technique in operable cases."

Clerf [14] in 1927 reviewed the damages to the larynx noted after radiation treatment.

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8. New, Gordon B., and Figi, Frederick A.: Treatment of Carcinoma of the Larynx, Surg., Gynec. & Obst. **62**:420-423, 1936.

9. Colledge, L.: Fourteen Cases of Total Laryngectomy for Intrinsic Cancer of the Larynx, J. Laryng. & Otol. **41**:19-27, 1926.

10. Fraser, J. S., and Watson, D.: Notes on Fourteen Cases of Intrinsic Cancer of the Larynx, with Lantern Demonstration of Pathological Anatomy, J. Laryng. & Otol. **39**:79-89, 1924.

11. Finzi, N. S., and Harmer, D.: Radium Treatment of Intrinsic Carcinoma of the Larynx, Brit. M. J. **2**:886-889, 1928.

12. Graham, H. B.: Window Resection of the Larynx for Treatment of Carcinoma, Tr. Am. Laryng., Rhin. & Otol. Soc. **34**:458-460, 1928.

13. Pancoast, H. K.: Radiation Treatment of Carcinoma of the Larynx, Am. J. Roentgenol. **12**:217-219, 1924.

14. Clerf, L. H.: Laryngeal Complications of Irradiation, Tr. Am. Laryng. A. **49**:99-112, 1927.

Escat, Ducuing and Rigaud,[15] of Brussels, reported in 1930 the results of treatment of 150 patients with carcinoma of the larynx. They stated that "with radium as a sole agent of treatment the results are truly bad in the present state of the technic used."

Stewart Harrison[16] reported in 1932 on the roentgen treatment of 15 patients with intrinsic carcinoma. Six were well for three and one-half years. He stated, however, that he still considered surgery as the method of choice.

Coutard[17] in 1932 outlined his technic of treatment and the results obtained. A five year cure was obtained in 13 of 60 cases (21 per cent) and a nine year cure in 4 of 8 cases.

William Harris[18] in 1935 in an article on protracted roentgen therapy stated: "At present it is probably safer to operate on the true cord tumors whenever possible."

T. J. Harris[19] reviewed the literature on carcinoma of the larynx and stated as his conclusion: "If operation is indicated, radium is not substituted."

Imperatori[20] in 1926 reported on 30 cases of extensive carcinoma. He stated: "The cure of cancer of the throat in all but exceptional cases would not seem to be attained through the use of radium."

Hinojar[21] found two years to be the average length of life of patients with carcinoma of the larynx, with and without treatment, except in cases in which thyrotomy was performed at an early stage.

Tilley[22] reported 3 cases of late "recurrence." One patient with carcinoma of the right vocal cord showed carcinoma of the left vocal cord thirteen years after operation; a patient with carcinoma of the right vocal cord showed a recurrence after nearly seventeen years and a patient with carcinoma of the left vocal cord showed a recurrence eleven years later in the operative scar.

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15. Escat, Ducuing and Rigaud: Résultats obtenus dans le traitement de 150 cancers du larynx par les radiations, Cancer, Bruxelles **7**:121-126, 1930.

16. Harrison, R. Stewart: Malignant Disease of the Larynx and Pharynx, J. Laryng. & Otol. **47**:725-755, 1932.

17. Coutard, H.: Roentgen Therapy of Epitheliomas of the Tonsillar Region, Hypopharynx and Larynx from 1920 to 1926, Am. J. Roentgenol. **28**:313-331, 1932.

18. Harris, William: Neoplasms of the Oral and Upper Respiratory Tracts Treated by Protracted Roentgen Therapy, Am. J. Roentgenol. **34**:482-491, 1935.

19. Harris, T. J.: Is Radium a Cure for Cancer of the Larynx? An Inquiry Based on a Study of the End-Results, Arch. Otolaryng. **5**:303-308 (April) 1927.

20. Imperatori, C. J.: Carcinoma of the Larynx and Laryngopharynx Treated with Radium: Analysis of Thirty Cases, Arch. Otolaryng. **4**:151-159 (Aug.) 1926.

21. Hinojar, A.: Little Difference in Length of Survival of Subjects with Treatment and Without, Cancer, Bruxelles **7**:138-144, 1930.

22. Tilley, H.: Late Recurrences of Cancer of the Larynx Following Successful Operations by Laryngofissure, J. Laryng. & Otol. **46**:241-247, 1931.

The summary of the literature seems to bring out the following points:

1. Operation in an early stage of growth is preferable to radiation treatment.

2. By means of laryngofissure a high percentage of cancers in the early stage can be successfully treated. The motility of the cord is an important factor in determining the type of operation. If at operation a growth is found to be larger than it was expected to be, the type of operation must be changed; in other words, the surgeon should not set out to perform any particular type of operation. It is also the general opinion that there are definite fields for laryngofissure, hemilaryngectomy and laryngectomy.

The following report is of 26 consecutive cases of carcinoma of the larynx in which operation was performed at the Johns Hopkins Hospital between 1912 and 1932. A period of five years has elapsed since the last patient underwent treatment. It is felt that that period gave ample time for the development of a local recurrence. Before operation all the patients were given a complete physical examination. A Wassermann test was made, the chest was studied roentgenographically and the teeth were examined. The growth in all cases was a squamous cell carcinoma, except in case 19, in which it was a teratoma. In all cases postoperative irradiation was given through the courtesy of Dr. C. F. Burnam, of the Howard A. Kelly Hospital. Patients showing an early stage of growth were given local or general anesthesia, and those showing a later stage of growth were given tri-bromethanol in amylene hydrate. An electric cautery was used in some cases to remove a growth in the early stages. The usual operative technic of thyrotomy and laryngectomy (one stage) was used. The patient's head was lowered when the larynx was opened or when it was removed, so as to prevent aspiration of blood. All patients were white men. Although Baltimore has a large Negro population and cancer of the larynx is frequently seen in members of this race, none with an operable growth was treated during this period.

There were 16 thyrotomies or partial resections, 10 laryngectomies, 1 local operation and 1 incomplete operation. Following the 16 thyrotomies there were 2 (cases 2 and 5) deaths (12.5 per cent). Three patients (cases 6, 8 and 16) could not be traced after they left the hospital. They probably died, as no news is bad news in these cases. Seven others (cases 1, 3, 9, 11, 12, 14 and 25) are known to have died; 3 patients (cases 17, 21 and 22) are alive and well, and 1 patient (case 20) is alive, but he has a recurrence in the glands of the neck nine years after operation.

A more detailed report of the 7 patients who survived operation but who have since died follows:

CASE 1.—A 50 year old man died five years after operation as a result of carcinoma of the base of the tongue.

CASE 3.—A 50 year old man died as a result of carcinoma of the pyriform sinus seventeen years after operation.

CASE 9.—A 62 year old man had a recurrence in one year. The vocal cord was fixed, and he should have had partial or total laryngectomy instead of thyrotomy.

CASE 11.—A 66 year old man died of carcinoma of the lung thirteen years after operation.

CASE 12.—A 45 year old man with fixation of the left vocal cord promptly showed a recurrence or continuation and died after laryngectomy. This patient should have been treated not by thyrotomy but rather by partial or total laryngectomy at the first operation.

CASE 14.—A 67 year old man died eight years after operation with carcinoma of the base of the tongue.

CASE 25.—A 43 year old man with a growth located posteriorly on the right vocal cord, with free movement, showed a recurrence in one year and died after laryngectomy.

Comment.—It is felt that in cases 9 and 12, in which operation was performed in 1921 (cord fixed by growth) the condition was not suitable for thyrotomy. Probably the entire larynx should have been removed at the time of operation. However, it is considered that in cases 1, 3, 11 and 14 the condition was suitable for operation, although death was caused by carcinoma of other parts, five, seventeen, thirteen and eight years, respectively, later, there was no reason to believe that the laryngeal growth was not completely removed at operation, as there was no local recurrence.

One patient (case 25) showed a condition suitable for hemilaryngectomy, but a sinus rapidly developed in the operative incision, as it did in all the cases in which there was an early recurrence. Three patients (cases 17, 21 and 22) are alive and well ten, nine and nine years, respectively, after operation.

A five year cure was obtained in 8 of 13 cases (61+ per cent), yet all the patients who survived operation and have since died showed carcinoma either as a recurrence or in some other part. The youngest patient who underwent thyrotomy (case 5) was 28 years old, and the oldest (case 17) was 77.

Among the 10 patients on whom laryngectomy was performed there were 3 postoperative deaths (33.3 per cent; cases 4, 7 and 25); 2 patients are alive and well (cases 13 and 26), fourteen and five years, respectively, after operation. One patient (case 10) was alive and well when last heard from, four years after operation (performed in 1920). Another patient (case 18) could not be traced. In cases 12 and 25 there was a recurrence following thyrotomy, and both patients died after laryngectomy. One patient (case 12) died six months later, from carcinoma of the glands of the neck. In case 25 postoperative death occurred. Another patient (case 15) died three months after operation, from pneumonia.

In case 24 death due to carcinoma of the glands of the neck occurred two years after laryngectomy was performed. The ages of the patients on whom laryngectomy was performed were as follows: 1 was in the third decade, 4 in the fourth decade, 2 in the fifth decade and 3 in the sixth decade. All the known dead in this

group died either from the operation or from carcinoma, except 1 patient (case 15), who died of pneumonia three months after operation.

CASE 23.—A 63 year old man had a small carcinoma removed from the vocal cord through a direct laryngoscope. He has remained well for eight years. This method is not recommended, but the patient has had no evidence of recurrence in the larynx.

CASE 19.—A 70 year old man with a large growth in the larynx had it partially removed through a laryngoscope after refusing laryngectomy. Section showed that the growth was a teratoma. The patient died one year later.

SUMMARY AND CONCLUSIONS

Four of the 26 patients operated on in the period between 1912 and 1932 could not be followed after they left the hospital. Of the 22 others, 7 are alive, including 1 patient (case 20) who had a recurrence in the glands of the neck nine years after laryngofissure. One patient (case 10) was alive and well four years after laryngectomy but could not be followed later.

Of the 13 patients on whom laryngofissure was performed and who could be traced, 3 are alive and well, and 1 is alive, with recurrence in the glands of the neck. In all the cases in which a recurrence developed there was a fistula in the operative scar. The 2 patients with fixation of the vocal cord were not helped by laryngofissure, both showing early recurrence. This was also true of case 25, in which the growth was located posteriorly on the vocal cord. Four patients who did not show a recurrence died of carcinoma of other parts from five to seventeen years after operation.

Two of the 8 traced patients who underwent laryngectomy are alive and well. Laryngectomy was performed on 2 patients who showed recurrence after laryngofissure, with little benefit, 1 patient dying postoperatively and 1 dying of carcinoma of the glands of the neck. The known deaths in this group were due either to operation or to a recurrence which developed within two years. Case 15 was the only exception, death being due to pneumonia.

True results of operations for cancer of the larynx are best learned from a careful study of the late results, which include the entire life of the patient.

It is desirable when possible to preserve the voice and the larynx, but this desire should not influence the operator when there is any doubt that the entire growth can be removed except by laryngectomy.

Although all the patients received postoperative radiation and some received extremely large doses, this did not prevent the development of recurrences or of new growths in adjacent parts.

All the patients who survived the operation and are known to have died (except 1, case 15) died of cancer either as a recurrence or in some other part of the body.

The only cases in which a "cure" was obtained were those in which careful operative removal of the entire tumor was carried out, followed by radiation treatment.

The danger of hoarseness and the imperative need for early operation must continue to be stressed both to the members of the medical profession and to the laity, as early diagnosis and operation are necessary for the successful treatment of cancer of the larynx.

ABSTRACT OF DISCUSSION

Dr. Gabriel Tucker, Philadelphia: Dr. Broyles' paper would lead one to believe that the surgical treatment of cancer of the larynx presents a sad picture. Dr. Broyles' patients were carefully and properly selected for surgical treatment, but I believe that he expects too much from his operative treatment of the larynx when he considers that an instance in which a patient dies of cancer in some other portion of the body over five years after the surgical treatment of cancer of the larynx can be counted as representing a failure of his method of surgical treatment. Laryngologists are agreed that the most important consideration in the surgical treatment of cancer of the larynx is early diagnosis and that laryngeal cancer treated by surgical intervention should be carefully selected as to location and extent of the lesion. Numerous series of cases have been reported in which the percentage of surgical cure over a five year period ran as high as from 70 to 85.

In properly selected instances of early cancer laryngofissure has given a particularly high percentage of cures. In my review of a series of 200 consecutive cases from the bronchoscopic clinics of the University of Pennsylvania I found that in 70 per cent the condition was probably of intrinsic origin and, had it been diagnosed early enough, would have been amenable to surgical therapy. When they were examined, however, only 56 per cent of the patients were recommended for surgical treatment because of the extent of the lesion. Six patients refused operation, 58 were treated by laryngofissure, 31 by total laryngectomy and 17 by partial laryngectomy with postoperative irradiation. Partial laryngectomy was done either because the patient refused total laryngectomy or because it was considered that the general condition contraindicated total laryngectomy. I believe that with present day methods of early diagnosis by means of direct laryngoscopic examination and biopsy, proper selection of cases with regard to location and extent of the lesion and the treatment of cancer of the larynx by laryngofissure, partial laryngectomy or total laryngectomy, well over 70 per cent of the patients can be permanently cured of laryngeal cancer. The surgeon who operates on a patient for cancer of the larynx undertakes the responsibility of surgical cure, and he should keep the patient under personal observation when it is at all possible. If there is no reappearance of cancer at the site of operation within a three year period it is possible that the subsequent occurrence of cancer should be considered as a new growth rather than as a recurrence. There is no operation or other treatment for cancer of the larynx at the present time that will prevent the development of cancer at some other location in the body. The treatment of laryngeal cancer by any form of irradiation does not compare in favorable end-results with its treatment by conservative surgical methods.

Dr. Murdock S. Equen, Atlanta, Ga.: Entirely too many patients come to the laryngologist with late cancer after the vocal cord is fixed, so that they can hardly speak. This is due partly to the laxity of the laity and partly to the

carelessness of physicians. Not enough attention is paid to hoarseness, and it is only by continually calling attention to the importance of early diagnosis that better results are to be obtained.

The late Dr. Robert C. Lynch of New Orleans devised a satisfactory technic for the treatment of early cancer of the larynx in which the affected vocal cord is freely movable. By this method the patient's head is suspended, and the operator works under direct vision through the natural channels. He grasps the growth with long-handled forceps in one hand, brings it toward the midline and with a long slender knife in the other hand dissects the tumor widely, going well into the normal portion of the cord. He then cauterizes the base with surgical diathermy. I modified the knife used by Lynch so as to insert a new blade for each operation; this knife was presented some years ago. I feel that with the Lynch technic one can get as good results as with thyrotomy in the majority of cases in which the growth is in an early stage, though I still resort occasionally to laryngofissure.

Of course, if the growth extends beyond the true vocal cord and the cord is not freely movable, the Lynch technic is not applicable and total laryngectomy is the operation of choice. This procedure is not as mutilating as some have pictured it. These patients seem to get along satisfactorily; some of them even enjoy using the artificial larynx. One man told me that no one had ever paid much attention to him before but since his operation if he goes to a soda fountain and orders a drink, he soon has a big crowd around him.

The scientists of the Bell Telephone Company are working on an artificial larynx which will give three tones. The tone of the one now used is metallic. When the new one is perfected, it will take away some disadvantages of the artificial larynx.

The mortality from total laryngectomy used to be about 100 per cent. By meticulous postoperative care the mortality has been brought within reasonable limits, and it can be further reduced. The problem concerns the management of feeding and of keeping the tracheobronchial tree free from secretions. The attempt was made to keep these patients alive by intravenous and rectal feeding but this could not be done until the wound was healed. By the use of the nasal tube, which can be worn for several weeks, the nutrition can be kept up satisfactorily. The secretions from the lung can be aspirated with the tonsil suction machine through a rubber catheter. Frequently the wound breaks down and the secretions run directly into the trachea and cause pneumonia and death. To avoid this complication the secretions must be kept out of the trachea by constant aspiration. It is purely a problem of nursing. I think the problem presented by surgical intervention is not nearly so great as that of nursing.

In conclusion, I think that surgical intervention offers the best opportunity to a person with cancer of the larynx, because radium and roentgen rays have not so far proved of sufficient value.

Dr. Gordon B. New, Rochester, Minn.: Surgical intervention along with surgical diathermy offers the best chance of ridding the patient of carcinoma of the larynx. Roentgen radiation and radium have a definite place in the treatment of malignant disease in this location. I do not feel, however, that they should be used for conditions from which there is a high incidence of recovery remaining after treatment by surgical removal and diathermy.

It would be instructive in the cases reported to know the duration of the disease and the activity of the growth as related to the type of operation employed in cases in which the operation was thyrotomy and laryngectomy. It is generally known that a rapidly growing tumor requires more radical treatment than

a slowly growing one. The location of the tumor also must be a factor in determining just how it should be taken care of. Perforation of the cartilage of the larynx means that a conservative operation is useless.

From the standpoint of methods of removal, malignant growths of the larynx may be divided into three groups: (1) supraglottic tumor, (2) intralaryngeal tumor, including subglottic growths, and (3) laryngopharyngeal tumor, arising in the pyriform fossa and the postcricoid region. Many of these growths, however, may involve two, or possibly all, of these regions, so that definite anatomic divisions cannot be made.

Tumor of the epiglottis of a low grade of malignancy in which the lesion is definitely limited to the epiglottis is best taken care of by performing preliminary tracheotomy. Then, by means of a Lynch suspension apparatus with a flat spatula, the growth may be removed under direct vision, a protected diathermy point being used. The use of this particular method, however, should be limited to cases in which there is no question that the growth can be removed in this manner. In cases in which the growth extends laterally to the aryepiglottic fold and the arytenoid region, subhyoid or lateral pharyngotomy is a better procedure. Whether one performs pharyngotomy through the thyrohyoid membrane after division and retraction of the hyoid bone or whether one performs lateral pharyngotomy must depend on the situation and extent of the lesion. In this way wide exposure can be made and as radical an excision performed as is necessary. In these cases a window may be removed from the thyroid cartilage on the uninvolved side; in this way sufficient exposure can be obtained, and the growth on the opposite side of the larynx can be destroyed with surgical diathermy through this opening. The transmandibular and translingual exposure as used by Trotter to remove an epiglottic growth has a very limited field. The other methods are far more efficient, and from them the patient will receive less general reaction.

A malignant lesion in the region of the vocal cords may be taken care of in any of four ways: 1. A growth involving the anterior two thirds of the vocal cord without fixation may be removed by thyrotomy, with excision of the growth and destruction of the base by surgical diathermy. 2. One involving the same region, but with fixation, may be removed by thyrotomy when it has not perforated through the thyroid cartilage, being removed along with the cartilage and the tissue being destroyed by surgical diathermy. A growth in the anterior commissure is best taken care of by division of the hyoid bone, the opening being made through the thyrohyoid membrane. In this way a careful examination can be made as to the exact extent of the growth. 3. In the treatment of a tumor of a low grade of malignancy, thyrotomy is performed, with removal of the diseased tissue, which frequently involves the anterior portion of both vocal cords and the anterior commissure. This method was suggested by Jackson and is very effective in the care of tumors of a low grade of malignancy. 4. In the treatment of a more extensive tumor with fixation involving the anterior commissure and the false cords, laryngectomy is done. Preliminary tracheotomy is done first, and then the larynx is removed from below.

The operative mortality reported by Dr. Broyles in 26 cases over a nineteen year period is not a fair estimate of the present day operative mortality. He reported 33 per cent operative mortality in laryngectomy and 12.5 per cent in thyrotomy. I recently reported a series of 135 consecutive instances of epithelioma of the larynx—70 thyrotomies were performed, with no death resulting from operation, and 65 laryngectomies, with one death.

The end-results must depend on the selection of treatment for the individual case, and this must be determined in most instances at the time of the operation. A freshly frozen section is made to determine the activity of the growth; the lesion is carefully examined, and the type of operation that seems best suited to the particular case is used. Surgical diathermy or actual cautery has been found to be a great aid in the treatment of malignant disease of the larynx. Dr. Waugh and I recently reported a study in which it was found that of a group of 107 patients who could be traced, 69, or 64.5 per cent, obtained five year cures; 53, seven year cures; 27, ten year cures; 8, fifteen year cures, and 3 twenty year cures. Fifty-six patients were still living when the paper was written. The average time which had elapsed since operation on the 56 patients was nine years and fourth months; the shortest time, five years, and the longest time, twenty-four years.

Dr. Louis H. Clerf, Philadelphia: I am particularly interested in Dr. Broyles' paper because of the question of late recurrence of carcinoma. When can a patient operated on for carcinoma be considered cured? Should one employ the term cured to designate that there is freedom from recurrence? Statistics are relative. The cases in which one is "unable to trace" the patient are disconcerting from a statistical standpoint. Recently I learned that a patient on whom I had operated for squamous carcinoma of the larynx eleven years previously had died of squamous carcinoma of the lung. Another patient, operated on almost ten years ago, has acquired a pulmonary lesion, presumably a malignant growth, which is producing bronchial compression. Are these instances of recurrence, or are the neoplasms primary? While this question is of more than statistical interest the fact remains that recurrence many years after removal of the primary growth should in no way interfere with the decision to operate early and to remove the carcinoma in those cases in which the condition is deemed suitable for operation. Perhaps one had better employ the term well, as suggested by the late Dr. McCrae, instead of cured.

Dr. E. Lee Myers, St. Louis: It is with considerable temerity that I appear on this program with such a small series of cases, but I have been considerably worried about my method of procedure in cancer of the larynx, and I am still more worried after having had an experience which I shall describe. In 1910, in a well selected case of a small growth in a larynx, Dr. N. A. Goldstein, with whom I was associated, chose to do a laryngofissure. The patient died later with widespread cancer. When I started for myself, I naturally thought that laryngofissure ought to be tried, and I did this operation in two or three cases. Radium could be had in 1912, but the patient had to be sent to another city, Belleville, for cross-firing with radium. This was done. The patients died of metastases. I chose recently a selected group of patients with small growths with no glandular involvement, and in collaboration with a surgeon I operated in about 11 cases. In every one of these cases the condition was ideal for laryngectomy. All the patients have died with metastases. One patient, not of this group, was sent to me for a laryngectomy, but he refused it. It has been four years since biopsy was performed. He has been under continuous observation and has done better than any of the other 11. Why?

Dr. Edwin N. Broyles, Baltimore: I cannot answer all the questions that were asked. I do not grade the malignancy of the tumors as 1, 2, 3 and 4, but it has been my impression from the cases I have studied that the earlier the decade of life in which the cancer starts, the more malignant the tumor seems to be.

If a cancer starts late in the seventh decade, I feel that I can treat it conservatively, whereas if it starts in the third or the fourth decade I should treat it radically.

I am sorry the picture that I painted was so bad, but I wanted to describe the true happenings to these patients over a long period. I hope to be able to follow up until I get a final group together and then to report the final outcome in all the 26 cases.

FOLLOW-UP OF PATIENTS EIGHT MONTHS AFTER TONSILLECTOMY

HAROLD D. SMITH, M.D.
BOSTON

In the fall of 1934 I became interested as to whether all the 2,200 tonsillectomies performed at the Massachusetts Eye and Ear Infirmary each year were really indicated. On the suggestion of Dr. Harris P. Mosher I correlated the history which led to tonsillectomy with the pathologic diagnosis in 104 cases of routine adenoidectomy and tonsillectomy. The article giving the results of my observations was published in the April 1935 number of the ARCHIVES OF OTOLARYNGOLOGY [1] and will be briefly quoted from later.

In the present paper I shall present, first, a correlation of the history which led to tonsillectomy, the pathologic diagnosis of the tonsil and the subsequent clinical course over a period of eight months. Second, the so-called immediate results of tonsillectomy, as noted in this study, will be compared with the less favorable results after a period of years, as shown by the figures of Dr. Albert D. Kaiser.

The correlation between the history which induced the patient to be operated on and the pathologic changes in the tonsils is summarized in chart 1. I quote from the article mentioned previously.

> As was to be expected, the largest group of patients, 45, or 43.26 per cent, complained of recurrent sore throat and colds. Of this number, 39, or 86⅔ per cent, showed chronic tonsillar infection. The remainder were normal.
>
> Seventeen patients, or 16.34 per cent, underwent tonsillectomy on account of discharging ears, repeated earache or some other aural difficulty. The tonsils of only 58.86 per cent of these patients showed evidence of infection. In the 8 patients, or 7.69 per cent, whose chief complaint was nasal obstruction there was an incidence of tonsillar infection of 62.5 per cent. Likewise, the 8 patients for whom no history was given on the clinical record had an incidence of tonsillar infection of 62.5 per cent.
>
> The next group, 5.67 per cent of the whole, was made up of those who had been referred from the schools because of large tonsils. Of these children 66⅔ per cent had infected tonsils.
>
> The patients in whom I was particularly interested were those whose tonsils were considered foci of infection for iritis, arthritis and so forth. This group was surprisingly small, being only 6.71 per cent of the total number, but 85.71 per cent showed infection in the tonsils.

1. Smith, H. D.: Microscopic Pathology of the Palatine Tonsil, Arch. Otolaryng. **21**:426 (April) 1935.

Curiously, the same number of patients complained of failure to gain weight or complained of "indigestion," namely, vague abdominal distress and belching. Each of these groups contained 3 patients, or 2.88 per cent of the total. Of the patients with "failure to gain weight," who seemed to have the more reasonable cause for tonsillectomy, only 33⅓ per cent had tonsils which showed infection, while of the patients with "indigestion" the tonsils of 66⅔ per cent showed infection. One reassuring thing is the fact that one of the "thin" patients who showed apparently normal tonsils has been thriving and gaining weight since the operation.

Two patients were operated on whose history recorded laryngitis, and 2 had occasional pain on either side of the throat. In these two groups there was infection in 50 per cent.

The other complaints consisted of recurrent cervical adenitis, "nerves" and enuresis with 1 patient in each group. All showed chronically infected tonsils, making the percentage 100.

During the past month (in the summer of 1935) one more step toward the answer was taken when 103 follow-up cards were sent out.

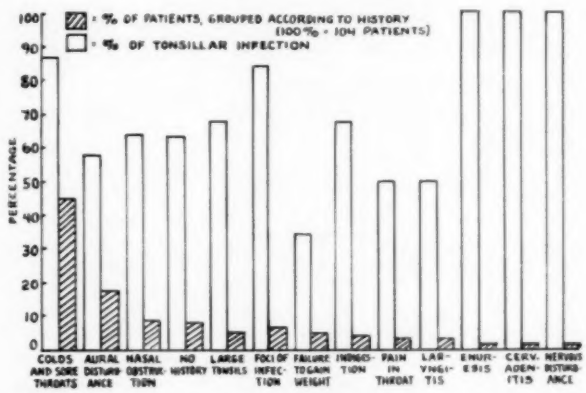

Chart 1.—Correlation between the complaint which induced the patient to be operated on and the pathologic condition of the tonsils noted at operation. Each of the shaded columns indicates the percentage of the entire group of patients (104) who were operated on because of the complaint indicated at the foot of the column. The unshaded column indicates the percentage of patients with the disease indicated who had tonsillar infection.

Now I have a correlation of the history leading up to the tonsillectomy, the pathologic diagnosis of the tonsil and the subsequent clinical course over a period of eight months. The eight months cover the fall, winter and spring of a New England year. Seven cards were unclaimed, and 61, or 59.22 per cent, were returned.

The general results are shown in chart 2.

After adenotonsillectomy 90.16 per cent of the patients had fewer colds while 9.84 per cent had the same number or more. Eighty-one and ninety-six hundredths per cent had fewer attacks of sore throat, but

18.04 per cent had the same number or more. My interest was stirred by the patients who had more attacks of sore throat following tonsillectomy than before, and a careful examination was carried out. The pharynges of these patients showed practically the same picture, that of engorged blood vessels lying on a beefy red mucous membrane, which was studded with lymphoid tissue. Lateral bands also seemed to be present in every case. This type of condition of the throat apparently corresponds with Dr. H. Archibald Nissen's type IV. He stated that "it is due to this focus (infected lymphoid tissue) that certain tonsillectomized patients continued to have their symptoms."

Eighty-eight and fifty-two hundredths per cent of the patients reported improved general health, while 11.48 per cent had noticed no change or expressed the opinion that they were worse. And 78.69

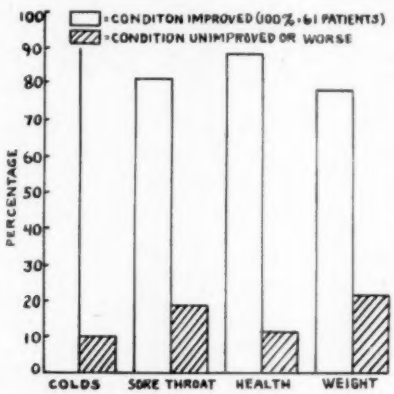

Chart 2.—Analysis of sixty-seven cases showing the results of the clinical and microscopic examination of the tonsils.

per cent stated that they had gained weight, the average of which was just over 5 pounds (2.3 Kg.). The remaining 21.31 per cent had neither gained nor lost.

Turning to the tonsils, I observed that the tonsils of 78.68 per cent of the patients showed pathologic changes and that those of 21.31 per cent appeared to be normal. The observations are shown in chart 3.

My consternation may be imagined when 84.61 per cent of the patients who had been regarded as having normal tonsils reported relief from their complaints and only 15.39 per cent said that they were no better or were worse. Only 75 per cent of the patients who had had tonsils showing a pathologic process gave a report of total relief. But 16.67 per cent reported partial relief, giving a total of 91.67 per cent having some abatement, at least, of their symptoms, while 8.33 per cent received no relief.

From these percentages it would appear that my diagnosis of normal tonsils was too high. It seems evident that the percentage of tonsils with pathologic changes should average between 85 and 90 per cent for a routine group of patients who give evidence by history and (or) by examination of the presence of infected tonsils instead of averaging about 78 per cent, as my first figures indicated. A recheck of the slides of sections of the tonsils of these patients seemed to bear out this conclusion. Using the criteria mentioned in the previous paper, I reached a percentage of 86 per cent of microscopically infected tonsils, with 14 per cent normal.

The final correlation is between the clinical symptoms which resulted in the tonsillectomy and the subsequent clinical course over a period of eight months. These results are summarized in chart 4.

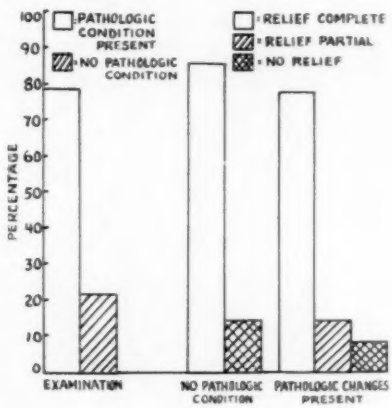

Chart 3.—Analysis of the clinical course of sixty-one patients, showing clinical changes after tonsillectomy in patients with and without pathologic changes in the tonsils.

Fifty-seven and thirty-seven hundredths per cent of the patients had had their tonsils and adenoids removed on account of recurrent colds and sore throat. Eighty per cent had complete relief from symptoms, and 17.42 per cent had partial relief. That is, those in the latter group had an abatement of either the colds or the sore throat but were not relieved of both. Two and fifty-eight hundredths per cent reported no relief.

All the patients suffering from some aural involvement stated that they were improved, but none had been cured. This group comprised 9.83 per cent of the patients. Of the 8.19 per cent who complained of nasal obstruction, 60 per cent received complete relief, 20 per cent partial relief and 20 per cent no relief. It was interesting that the 6.55

per cent who had had their tonsils removed as being foci of infection all claimed clinical improvement in their symptoms. Several of the patients said that they were completely cured, but examination did not bear out such a happy report. Of the 4.91 per cent of the patients who had no history recorded on their clinic records, 66.66 per cent reported improvement and 33.33 per cent partial improvement. The patients who had complained of indigestion divided the honors, with 50 per cent reporting cure and 50 per cent partial cure. However, the 2 persons, 3.27 per cent of the entire group, who had their tonsils removed on account of laryngitis were unanimous in saying that they had no relief, which was the first group in which there was 100 per cent failure. Likewise, the 1 patient who had pain in her throat said that

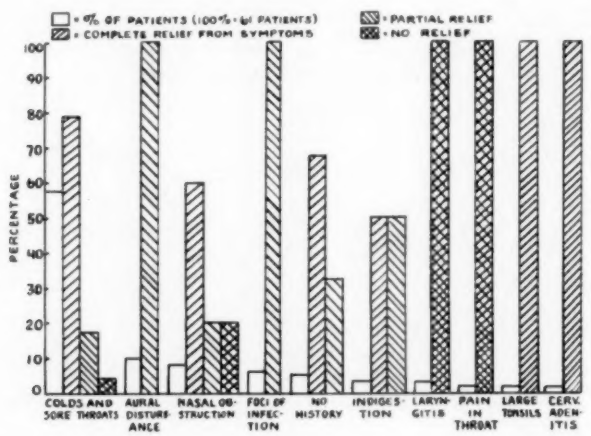

Chart 4.—Correlation between the clinical symptoms which led to tonsillectomy and the subsequent clinical course. The unshaded columns indicate the percentage of the entire group who had the symptom indicated at the foot of the column, and the shaded columns indicate the percentage of those presenting the symptom who obtained complete, partial or no relief, as indicated by the type of shading.

there was no improvement. The 1 patient with enlarged tonsils who replied had 100 per cent cure, as was to be expected. And the 1 patient with cervical adenitis stated that he was completely relieved of the symptoms.

COMMENT

It is hardly necessary to point out that the results given here cover only an eight month postoperative period and hence are essentially those immediately obtained. As Dr. Albert D. Kaiser brought out, it unfortunately seems to be true that the beneficial effects of adenoidectomy and tonsillectomy gradually decrease after an optimum period of about

one year. In this respect it is of value to compare Dr. Kaiser's figures with those obtained in this study.

Dr. Kaiser had a remarkable series of 2,200 children on whom he determined the incidence of infection before tonsillectomy and ten years after the operation. These results were checked against those obtained in an equal number of children who had not been operated on.

Some of Dr. Kaiser's figures which are applicable to this study are shown in chart 5. He reported improvement in the incidence of sore throat in 72.22 per cent of the tonsillectomized group, as against 12.19 per cent in the control group in whom tonsillectomy had not been performed.

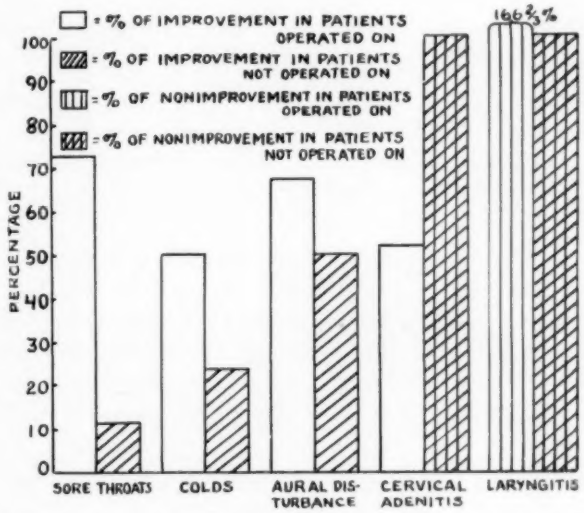

Chart 5.—Summary of Dr. Kaiser's data concerning 2,200 children followed over a period of ten years after tonsillectomy.

The incidence of colds was improved in only 50 per cent by operation, and the control group showed 24.39 per cent improvement. The results in patients with aural involvement were not so impressive. Only 66.66 per cent of the patients in whom operation was performed were improved, and 50 per cent of the control group showed improvement. The difference was marked in patients with cervical adenitis, however, with 53.33 per cent of the tonsillectomized children improved while there was no improvement in the control group. And with regard to laryngitis Dr. Kaiser even surpassed my 100 per cent failure, by showing an increase of 166.66 per cent in incidence of recurrent hoarseness in the tonsillectomized patients. The children in the control series showed an increase of only 100 per cent in the incidence of laryngitis.

SUMMARY

A correlation of the history leading to the tonsillectomy, the pathologic diagnosis of the tonsils and the subsequent clinical course over a period of eight months is presented.

From this study it appears that in a routine group of patients the percentage of persons who give evidence by history and (or) clinical examination of infected tonsils should be between 85 and 90. In this series the microscopic examination of the slides revealed 86 per cent to have had tonsils with a pathologic change and 14 per cent to have had normal tonsils.

Tonsillectomy and adenoidectomy have proved beneficial in cases in which a focus of infection was suspected in the tonsils and in which there was a history of recurrent colds and sore throat, aural involvement, nasal obstruction, cervical adenitis and enlarged tonsils. The procedure seems to be of questionable value in cases of indigestion, laryngitis and pain in the throat.

The so-called immediate results of tonsillectomy, as noted in this study, are compared with the less favorable results after a period of ten years, as shown by the figures of Dr. Albert D. Kaiser. Dr. Kaiser reported 72.22 per cent improvement in the incidence of sore throat in his patients on whom tonsillectomy was performed, and 12.19 per cent improvement in the controls, while 81.96 per cent of my patients reported improvement in the incidence of sore throat eight months postoperatively. There was a 50 per cent improvement in the incidence of colds in Dr. Kaiser's patients and 24.39 per cent in his control children, and 90.16 per cent of the patients in my series reported improvement. Only 66.66 per cent of Dr. Kaiser's children who had a tonsillectomy showed improvement in the incidence of aural involvement. All the patients of the present series claimed improvement, i. e., 100 per cent. Likewise, 100 per cent of the patients in this series with cervical adenitis were improved, while there was improvement in only 53.33 per cent of Dr. Kaiser's patients on whom tonsillectomy was performed. However, those in the control group reported 100 per cent increase in the incidence of the complaint. There was failure in 100 per cent of the patients with laryngitis in my series, which corresponds to the increase of 100 per cent in the incidence of laryngitis among those in Dr. Kaiser's control group. His tonsillectomized patients showed an increase in the incidence of laryngitis of 166.66 per cent.

While I have no sympathy with the promiscuous "slaughter of the tonsil," I have become firmly convinced that such a term is not justifiable if the physician has taken the history carefully and has made a thorough clinical examination, which has revealed infected tonsils.

Clinical Notes

PRIMARY HEMANGIOMA OF THE NASAL BONE

HARRY NEIVERT, M.D., AND EDWIN B. BILCHICK, M.D., NEW YORK

Primary hemangioma of any bone is a rare condition. Primary hemangioma of the nasal bone, as far as we could determine, has never been reported. Hemangioma is a common type of tumor occurring in such locations as the liver, eyelid, orbit or tongue. It is composed of vascular spaces lined by endothelial cells, the spaces being occupied by blood cells and separated by a thin avascular connective tissue stroma. Primary hemangioma of the bone arises within the bony substance, as opposed to hemangioma which arises in another tissue and then invades the bone. The latter is occasionally found in the nasal cavity, invading the septum or the sinuses. The cause of hemangioma of the bone is unknown. Trauma does not seem to be a causative factor. The presence of cell rests in the vascular spaces of the bone may account for the growth.

The first report of roentgen examination of primary hemangioma of the bone was that of Hitzrot [1] in 1917. The tumor involved the upper end of the humerus. Cushing [2] removed a tumor of this nature from the parietal bone. Bucy and Capp [3] presented a most exhaustive and authoritative description of primary hemangioma of the bone. They reviewed the literature, summarized the cases reported up to 1930 and reported eight instances of primary hemangioma of the bone in various locations. A search through the literature rewarded us with reports of several instances of this type of tumor in some of the bones of the skull and face, but ours is the first case of primary hemangioma of the nasal bone on record.

The growth begins in the vascular spaces of the bone, enlarges by the formation of more and larger blood channels filled with red blood cells and lined by endothelial cells. It penetrates the cortex and causes some enlargement and swelling of the bone, but it does not go beyond the periosteum which is stretched over the bone. It is a benign tumor with no metastases. Some bony trabeculae are absorbed, and others are thickened in such a manner that the resultant appearance of numerous spicules of bone radiating from a common center at right angles to the plane of the bone, described as "honeycomb" or "sunburst," is characteristic of the lesion, and its roentgen appearance is pathognomonic.

REPORT OF A CASE

Miss M. R., a dressmaker aged 32, was admitted to the Presbyterian Hospital in New York on Feb. 18, 1935. Her chief complaint was of pain and swelling over the right side of the nose.

From the Otolaryngological Department of the Columbia University Presbyterian Hospital Medical Center, Dr. John D. Kernan, Director.

1. Hitzrot, J. M.: Hemangioma Cavernosum of Bone, Ann. Surg. **65**:476-482, 1917.

2. Cushing, H.: Surgical End Results in General, with a Case of Hemangioma of the Skull in Particular, Surg., Gynec. & Obst. **36**:303-308, 1923.

3. Bucy, P. C., and Capp, C. S.: Primary Hemangioma of Bone, with Special Reference to Roentgenologic Diagnosis, Am. J. Roentgenol. **23**:1-33, 1930.

History.—The patient had always been in good health prior to the onset of this condition. There was no history of injury or of operation. There had been no systemic or infectious disease. The family history was essentially unimportant.

Present Illness.—During the four months before examination the patient noticed gradually increasing pain over the right side of the nose, near the nasofrontal angle. The pain radiated to the right supra-orbital region. There was swelling over the right nasal bone, increasing in size. No epistaxis or nasal discharge was noted.

Physical Examination.—The patient was a well developed, well nourished white woman. General examination gave essentially negative results. The heart had a regular rhythm, with a slightly rapid rate. No murmurs were noted. The lungs were clear. The blood pressure was 125 systolic and 80 diastolic.

Fig. 1.—Roentgenogram of the right nasal bone taken before operation, showing radiating spicules of bone perpendicular to the bridge.

Otolaryngologic Examination.—The ears were essentially normal. The nasopharynx showed no growth, adenoids or discharge. The tonsils were cryptic, with reddened anterior pillars. The larynx was normal. Transillumination of the nasal accessory sinuses showed the frontal sinuses to be large and clear. The maxillary antrums were equally clear.

In the external nares a hard tender swelling was seen over the right nasal bone extending to the nasofrontal angle. No fluctuation could be elicited. The left nasal bone was normal. The mass measured about 2 cm. in diameter.

The internal nares gave negative results for bleeding and for the presence of a tumor mass. No discharge or ulceration could be noted. The right middle turbinate bone was somewhat granular. Examination with the nasopharyngoscope gave negative results. The Wassermann reaction of the blood on Feb. 20,

1935, was reported to be negative. Examination of the urine for albumin and sugar on Feb. 18, 1935, gave negative results. Roentgenograms of the nasal bones and sinuses were interpreted by Dr. Haig Kasabach as follows: "The frontal sinuses are multilocular. The cells on the lateral aspect are deeper and sharply outlined. The central portions are shallow, with localized sclerosis of the posterior group of cells. There is no evidence of sinusitis. The frontal, ethmoid and sphenoid sinuses are clear.

"Roentgenograms of the facial bones with special reference to the nasal bones, particularly in the lateral view, show numerous fine reticulated lines, most of which are perpendicular to the bridge of the nose. These lines are interspersed with fine radiolucent areas. There is no break in the contour of the bone. The lesion extends rather close to the tip anteriorly and near to the posterior margin of the nasal bones posteriorly. Because of the presence of these fine radiating spicules, the bone appears somewhat widened and thickened in some places.

Fig. 2.—Roentgenogram taken nine months after operation, showing almost complete disappearance of the lesion.

"The changes seen in the nasal bone could be produced either by a tumor or by infection. The absence of any break in the continuity of the outline of the bone excludes the possibility that the growth is a malignant tumor. A chronic infection is expected to produce a certain degree of thickening of the bone without a reticulated pattern, such as is present in this case. My first impression is that this is a benign tumor, probably a hemangioma. A cartilaginous tumor may produce a similar picture."

Operation was performed by one of us (H. N.) on Feb. 27, 1935, with the patient under anesthesia with tri-bromethanol in amylene hydrate and ether.

Pathologic Picture.—There was a bony tumor on the right nasal bone, near the nasofrontal angle. This tumor extended downward and into the nasal bone, so that on its removal a hole was left from the interior of the nose to the soft tissues on the outside of the nose. The tumor was spongelike.

Fig. 3.—Section stained with hematoxylin and eosin, showing bony trabeculae, large cavernous blood vessels, and spaces filled with erythrocytes lined by endothelium. Intercellular stroma is small in amount (× 150).

Fig. 4.—Section stained with hematoxylin and eosin, showing long, flat endothelial cells lining cavernous spaces (× 600).

Procedure.—An incision about 1 inch (2.5 cm.) long was made in the right nasofrontal angle directly over the tumor. The soft tissues were removed from the tumor by blunt dissection, and the tumor was removed piecemeal with curets. The wound, which extended directly into the right nasal cavity was packed with iodoform gauze and then sutured. The right nasal chamber was packed with gauze.

The postoperative reaction was slight, and recovery was uneventful. The external wound healed quickly. There was some edema of the right eye, which subsided in a few days. The sutures were removed. No infection was noted. The patient was discharged on March 6, 1935.

Pathologic Report.—Pathologic examination of the tumor was made by Dr. F. M. Smith.

Macroscopic Examination: "The specimen consists of seven irregular ragged ecchymotic bits of firm tissue containing bone and varying from 4 to 8 mm. in diameter. These are somewhat spongy, and all of them grit when cut with a knife."

Microscopic Examination: "The bony trabeculae are composed of relatively normal bone. They are, however, widely separated from one another and are sur-

Fig. 5.—Photograph taken after operation, showing the inconspicuous scar over the right nasal bone. No deformity is present.

rounded by loose connective tissue. The spaces between the trabeculae are filled for the most part with extremely large blood vessels, which in some places show slight thickening of the endothelial lining. In a few places the trabeculae are somewhat thinned out.

"This is apparently hemangioma of the bone, which is a rather rare condition, especially in the nasal bone. It is most often seen in the vertebrae. Hemangioma of the bone is not as a rule a malignant metastasizing tumor."

Follow-Up.—On April 1, 1935, the interior of the nose was found to be normal. There was a slight swelling over the operative scar, with some tenderness.

Roentgenograms of the nasal bones taken on June 24, 1935, were interpreted as follows: "There is no apparent recurrence of the tumor."

Roentgenograms made on July 1, 1935, were interpreted as follows: "Part of the bone has been removed. The base of the nasal bone shows a number of extremely small honeycomb patterns."

Examination of the nose on July 5, 1935, and on Aug. 30, 1935, revealed a normal mucosa. Pain had disappeared. On Dec. 6, 1935, examination showed complete shrinkage of the swelling, with a slight residual irregularity over the scar.

On Jan. 17, 1936, roentgenograms of the femurs, the lumbar portion of the spine, the thoracic portion of the spine and the pelvis, taken to reveal any possible multiplicity of lesions, failed to show any tumor in these bones. Examination of the nose on Feb. 7, 1936, showed a healed inconspicuous scar. The nares were clean and clear. When the patient was seen on March 17, 1936, about a year after the operation, there was every indication of a cure.

Comment.—The lesion in the case reported here was a typical primary hemangioma of bone, diagnosed by roentgenograms, the diagnosis being confirmed by microscopic examination. Its interest lies in its rarity and its differentiation from sarcoma of the nasal bone. A sarcoma breaks through the periosteum and invades adjacent structures, whereas a hemangioma stretches the periosteum but does not go through it.[4] Sarcoma, while it shows bony spicules, does not show these to appear perpendicular to the plane of the bone on roentgen examination, nor do the spicules radiate from a common center, as do those in hemangioma.

The treatment of hemangioma of the bone is preferably surgical. If the tumor is accessible, removal of the lesion will effect a cure. Roentgen therapy has been shown to cause diminution and even disappearance of the lesion when carried out over a long period. The efficacy of radium therapy has not been determined.

SUMMARY

An instance of primary hemangioma of the nasal bone is described. This seems to be the first case of this condition reported in the literature.

Diagnosis was made by the pathognomonic appearance on roentgenograms of the radiating bony trabeculae and a honeycomb structure. It was confirmed by microscopic findings of bony trabeculae separated by numerous large vascular spaces lined by normal endothelial cells.

The tumor was removed surgically, with disappearance of symptoms and lack of deformity.

4. Bucy, P. C.: Pathology of Hemangioma of Bone, Am. J. Path. **5**:381-388, 1929.

MYIASIS OF THE LARYNX

ALDEN H. MILLER, M.D., LOS ANGELES

On Sept. 27, 1935, a Mexican girl of 6 years was sent to the laryngeal service of the Los Angeles County Hospital by a private physician, who had made the diagnosis of laryngeal diphtheria and had given 10,000 units of diphtheria antitoxin. On admission the patient gave the history of having acquired a cold five days previously, with a sore throat developing on the second day and dyspnea on the third day. She had had a high fever since the onset of the condition.

On examination the child seemed well nourished. She was breathing with difficulty and appeared ill but able to talk hoarsely. Her temperature was 103.6 F. Breathing was harsh, and there was a harsh, brassy cough. Examination of the nose demonstrated apparent atrophic rhinitis, but no ulceration was present. The entire posterior pharyngeal wall, extending from the nasopharynx to the larynx,

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was necrotic and swollen. Just above the larynx on the postpharyngeal wall was a large black ulcerated area of necrotic tissue, from which many maggots could be seen escaping. Both tonsils were swollen and red, the right one being riddled with maggots. When the maggots were discovered the patient said that she had eaten partially spoiled fruit picked from the ground in an orchard without having first washed off the dirt.

All visible maggots were removed under direct laryngoscopic observation. During the next three days respiration was less labored; numerous maggots were coughed up from time to time, and a small hemoptysis occurred occasionally. On the patient's fifth day in the hospital a second direct laryngoscopy was done, and all visible maggots were removed from the posterior wall of the hypopharynx, the pyriform sinuses and the right tonsil. At that time the patient had a considerable hemorrhage from the throat and was given a transfusion of citrated blood. She continued to have numerous small hemorrhages throughout the night, with occasional periods of dyspnea. Early in the morning of her sixth day in the hospital the pulse steadily became slower and weaker and respirations were short and gasping, and she died.

Autopsy revealed ulceration of the right tonsil, the posterior wall of the pharynx, the hypopharynx and the left pyriform sinus, and erosion of the posterior ends of the vocal cords and of the mucous membrane in the subglottic area. There was a mass of antemortem blood clot in the larynx. Bloody matter, but no maggots, was found in the lungs, stomach and bowels. No ulceration was present in these organs.

The anatomic diagnosis was necrotic ulceration of the pharynx and the larynx due to maggots, hemorrhage into the larynx, asphyxia and bronchopneumonia.

This case is of interest because, while numerous instances of infestation of the nose and ear by screw worms have been reported in the United States, although they are more common in the tropics, a review of the literature reveals only one report of involvement of the throat—that reported by Crocket [1] in 1929—and none of the larynx. The condition of Dr. Crocket's patient resembled that reported here in that there was involvement of a tonsil, with many hemorrhages and that the condition terminated in death, probably from shock due to hemorrhage.

It is also of interest that atrophic rhinitis and probably atrophy of the pharynx were present in this case, as well as in most others reported, as it is held that maggots do not attack healthy human tissues. Atrophic rhinitis is usually found in all patients whose nose or sinuses are infested by screw worms.

This case and that reported by Dr. Crocket seem to indicate that the prognosis is poor if screw worms are found attacking the pharynx or larynx.

The treatment indicated seems to be early and complete removal of the screw worms, since in both cases death followed even after all maggots had been removed (none being found at autopsy).

The larvae of flies attacking the human body belong largely to the families Oestridae, Calliaphoridae and Sarcophagidae.[2] The first group feed usually on exudations and serum without extensive destruction of tissue. The most common member of this group is the sheep bot. The blow flies are the typical members of the Calliaphoridae and are the most dangerous to man. Members of this family

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1. Crocket, John A.: Death from Texas Screw Worm in the Throat (Compsomyia Macellaria), Eye, Ear, Nose & Throat Monthly **8**:309 (Aug.) 1929.

2. Hegner, R. W.; Root, F. M., and Augustine, D. L.: Animal Parasitology, with Special Reference to Man and the Domesticated Animals, New York, Century Company, 1929.

are the Calliaphora, Scynomyia, Lucilia, Phormia and Chrysomyia (the one found in this case). The Sarcophagia rarely attack the passages in the head but are found in the intestinal tract.

Identification of the fly larvae is usually made by microscopic study of sections of the posterior spiracles, which consist of three parts: 1. Surrounding the whole spiracle in some species there is a complete chitinous ring. 2. Within this are the slits, the actual apertures by which air enters the body of the larva. Typically there are three straight or sinuous slits, each divided into a number of smaller apertures by a series of chitinous bars. 3. There is usually a small round chitinized area, often perforated, called the "button." The Chrysomyia macellarea have three straight slits pointed downward and no button. The common house fly has three winding slits and a button. The greenbottle fly, which is also called the outdoor fly or sheep maggot, has three straight slits turned toward each other and downward and a button. These three are the maggots usually found infesting human beings in the United States.

PETROSITIS WITH OPERATION AND RECOVERY

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The past history of this patient, a boy of 9 years, is irrelevant except for a tendency to acidosis on slight provocation associated with a somewhat rapid pulse and vomiting. He had no previous operations except a tonsillectomy and adenoidectomy a few years before the onset of the condition present at the time of his admission.

The present illness started on July 10, 1934, at which time the patient acquired an abscess of the right ear, one day after swimming. The drum was incised at that time. I first saw the child about ten days later. Examination disclosed a profuse discharge from the middle ear, moderate sagging of the superoposterior wall of the canal and definite tenderness over the mastoid antrum. The temperature was 103 F., and he complained of severe pain back of the ear. The whole clinical picture definitely presented evidence of typical suppurative mastoiditis. The blood count showed 13,000 white blood cells, of which 75 per cent were polymorphonuclears, many containing toxic granules. The roentgenogram showed some breaking down in a well pneumatized mastoid. Dr. Meierhof, with whom I observed the child, agreed with me that operation was indicated. A mastoidectomy done the next day (July 19) revealed pus and necrotic bone throughout an exceptionally pneumatic mastoid process. Indeed, the pneumatization was so extensive that I suggested to my confreres the possibility of subsequent petrositis. The organism cultured from the pus from the mastoid process was reported to be Streptococcus haemolyticus-beta.

The postoperative course was uneventful except for low grade fever, which was associated with somewhat more pain than is usually noticed in the ordinary course after mastoidectomy. The temperature at no time reached as high as 101 F. However, ten days after operation, the temperature had become normal and the pain had vanished; so the child was sent home. The wound was fairly dry, but it was noted that there was a profuse discharge from the middle ear. A few days afterward there began to be severe pain, more marked at night, located behind the right eye on the same side as the mastoidectomy. The temperature rose gradually to 102 F. and then went back to normal.

On August 21, three weeks after operation, the temperature again began to rise, reaching about 102 F. in three days. Coincident with this there was a definite change in the type of pain, which I deemed of importance. Instead of the pain back of the eye, worse at night, there occurred a more or less constant throbbing pain in the right frontal region. This type of pain, plus the rise in temperature, led me to make a diagnosis of epidural abscess due to rupture (through the cortex of the petrous pyramid) of suppurative petrositis. This diagnosis was strengthened by the decalcification of the tip of the right petrous pyramid, as shown by roentgenograms. The blood count showed 20,000 white cells, of which 75 per cent were polymorphonuclears. Of these 25 per cent were staff cells. Many contained toxic granules. The hemoglobin content was 76 per cent. General physical and neurologic examinations gave negative results. Dr. Friesner and Dr. Maybaum agreed that operation was indicated.

On that day, August 24, the patient was operated on. The mastoid cavity was reopened, but nothing was found except normal granulation tissue. No diseased tract could be located. A wide exposure of the sinus, cerebellum and cerebrum revealed no pathologic condition. I then removed a large piece of the squama, about 2 inches (5 cm.) in diameter, above the petrous pyramid, which enabled me to lift the brain away from the superior surface of the petrous pyramid. Although the exposure thus obtained was adequate, it could have been rendered better by a preliminary ventricular aspiration. A piece of rubber dam was pushed along the surface of the pyramid past the arcuate eminence. At the region of the apex, about ½ drachm (1.84 cc.) of thick pus was encountered. At that spot, a bent probe was felt to come in contact with necrotic bone on the surface of the pyramid. Culture of the pus showed Str. haemolyticus-beta. The rubber dam drain was then pushed abong the surface of the petrous pyramid to the perforation. A gauze drain was placed beneath the rubber dam. The mastoid cavity was packed open. No sutures were inserted.

When the patient was put back to bed, the temperature was found to have risen to 104 F. During the next few days it gradually dropped to normal. The throbbing pain, on which I had laid stress, promptly disappeared (although there was an occasional slight pain for a few days). The pain in the eyes and teeth took about two weeks to disappear.

The blood count a week after operation showed 12,000 white cells of which 60 per cent were polymorphonuclears. The hemoglobin content was 55 per cent. The deep packings down to the perforation were left in for five weeks and then were removed fairly easily, but on the next day there was a reaction, consisting of a rise in temperature to 101 F., pain in the eye, headache and vomiting. This subsided in twenty-four hours, and after that recovery was uncomplicated except for a moderate discharge of thick material for eighteen months before the ear became dry. At the time of writing the patient can hear a moderately loud whisper. The defect in the skull has shown little evidence of regeneration of the bone. The ear is dry.

The purpose of reporting this case is, first, to add another case to the literature, second, to show the best method, in my opinion, of approaching this type of case and, third, to stress the significance of the change in the type of pain when the pus breaks through the cortex, that is, the abrupt change from the characteristic nocturnal ocular pain to a throbbing steady pain in the frontal area. This change is definite and easily noticeable, and to my mind, in conjunction with other clinical symptoms it is a distinct indication for operative exposure of the apex of the petrous pyramid.

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Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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ACUTE AND CHRONIC OTITIS MEDIA, SINUS THROMBOSIS AND SUPPURATION OF THE PETROUS PYRAMID

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NEW YORK

In the review of the literature covering 1935 the great amount of work from the Russian otolaryngologists is striking. These observers seem deeply concerned with fundamentals, and they appear to be passing through a phase of study through which the rest of the otologic world has gone and on which it has reached accepted conclusions. With the Russian observers these matters are now in a stage of acute controversy. Perhaps in the end their reexamination of fundamentals may serve for a revaluation and also serve the purpose of showing the necessity for a revision of previously accepted ideas. To date, however, no noteworthy new concept has been developed.

Furthermore, the current literature gives evidence that the topic of petrositis has lost its strangeness and novelty. It is accepted as established in otology. The American students of the lesions of the petrosal pyramid collaborated in the presentation of a symposium at the American Otological Society in 1935, and scientific data and clinical observations were collected and assembled for further study and elucidation.

STUDIES IN FUNDAMENTALS

It has been held that the membrana flaccida differs from the membrana tensa in that the former has no substantia propria. Marx [1] made a series of histologic examinations and found that in half the cases which he studied a definite substantia propria could be demonstrated in Shrapnell's membrane. This finding must be interpreted in the light of other histologic studies. The radiating and circular fibers in the pars tensa do not extend above the height of the annulus tympanicus, into which they are inserted. These fibers are absent in the pars flaccida, and while a substantia propria can be demonstrated in some instances

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1. Marx, R.: Ueber den Bau der shrapnellschen Membran, Beitr. z. prakt. u. theoret. Hals-, Nasen- u. Ohrenh. **31**:431, 1935.

in Shrapnell's membrane, it is not to be understood that this in any way resembles the distribution of the middle layer of the drum in the pars tensa.

It is common usage to speak of the lining of the tympanic cavity as a mucous membrane. Studies are on record in which this lining membrane has been found to contain, in some places, secreting glands or goblet cells. Wittmaack,[2] however, has since shown that these are evidences of a pathologic type of mucous membrane which is the end-result of the hyperplastic type of infantile otitis. His statement is borne out by Gioacchini,[3] whose histologic studies demonstrated no glands in the lining membrane of the middle ear. He stated that it would be inconsistent with the physiologic function of the middle ear for glands to be present in its lining membrane. I feel that it would be advisable to discard the term mucous membrane not only in designating the lining of the tympanic cavity but also for that which covers the walls of the air-containing cells in the mastoid process. Confusion of scientific thinking might thus be lessened.

Ernyei and Bacsich [4] conducted a series of investigations on the nerves and ganglions of the middle ears of rats and guinea-pigs. They found that besides being supplied by branches from the fifth, seventh and ninth cranial nerves, the tympanic cavity has a sympathetic nerve supply which comes from the nervus jugularis, passing through the jugular ganglion to the middle ear, where it takes part in the formation of the tympanic plexus. The course of this nerve was hitherto unknown.

The understanding of the importance of the process of pneumatization on the course and type of subsequently developing processes in the middle ear and the mastoid process continues to be augmented from the clinical standpoint. Stix [5] found in a series of roentgenologic studies that if the process of pneumatization in children is disturbed there results a greater tendency to the production of protracted otorrhea. He found that the more normal the type of pneumatization the greater is the possibility that a coalescent mastoiditis can result and that surgical mastoiditis may develop. On the contrary, the greater the disturbance

2. Wittmaack, K., in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1926, vol. 12.

3. Gioacchini, R.: Sulla supposta presenza di glandole nella mucosa della cassa timpanica, Valsalva **11**:165, 1935.

4. Ernyei, I., and Bacsich, P.: Ein Beitrag zur Kenntnis der Nerven und Ganglien der Paukenhöhle bei Ratten und Meerschweinchen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **37**:276, 1935.

5. Stix, K. E.: Pneumatisationsverhältnisse des Wartzenfortsatzes bei verschiedenen Mittelohrerkrankungen grösserer Kinder, Beitr. z. prakt. u. theoret. Hals-, Nasen- u. Ohrenh. **31**:474, 1935.

to the process of pneumatization, the greater the tendency to produce a lesion of the middle ear, which inherently tends to chronicity.

ACUTE PURULENT OTITIS MEDIA

Pinaud [6] reports 3 cases in which acute purulent otitis media followed the use of postnasal packing. This observation is not new, but his advice to insert the finger into the posterior nasopharynx following a tamponade and to push the tampon away from the tubal ostium deserves consideration and adoption. The aeration of the eustachian tube is thus maintained, and a subsequent infection, if not obviated, at least is lessened.

To an old clinical phenomenon Asherson [7] gives a descriptive name. He states that in instances of acute purulent otitis in which the external auditory canal is mopped clean and then again fills rapidly in a short space of time, the refilling indicates that the discharge is coming from an overflow from the mastoid process. He terms this "the reservoir sign." I have observed this sign many times. It can be augmented by making digital pressure on the internal jugular vein along the anterior border of the sternocleidomastoid muscle during the observation. This procedure will result in an accentuation of the so-called reservoir sign.

Neumann [8] discusses the influence of bacterial flora on the course of an acute infection of the middle ear. In the majority of instances of acute purulent otitis media the course is influenced more by the type of pneumatization than by the bacteria which produce the lesion. The type III pneumococcus and the tubercle bacillus produce characteristic lesions. So do the other specific organisms, such as the diphtheria bacillus, Friedländer's bacillus, Actinomyces and Spirochaeta pallida. Neumann states that Streptococcus viridans and the influenzal bacillus produce symptoms which run a mild course. This is due to the mild local tissue reaction from these infecting organisms. The severe course of an infection of the middle ear is usually caused by a strain of Streptococcus haemolyticus, and this organism is solely responsible for the hemorrhagic type of otitis.

In conjunction with specific clinical pictures the work of Altmann [9] and Baum [10] is interesting. The former reports 7 cases of otomas-

6. Pinaud: Otites et mastoiditis provoquées par le tamponnement postérieur des fosses nasales, Rev. de laryng. **56**:106, 1935.

7. Asherson, N.: An Early Clinical Sign of Acute Mastoiditis, Lancet **1**:150, 1935.

8. Neumann, H.: Welchen Einfluss hat die Bakterienflora auf den Verlauf der akuten Mittelohrentzündung? Wien. klin. Wchnschr. **48**:275, 1935.

9. Altmann, F.: Zur Symptomatologie und Klinik der Friedländer-Otitis, Monatschr. f. Ohrenh. **69**:513, 1935.

10. Baum, H. L.: Pneumonic Otitis Media, Arch. Otolaryng. **21**:96 (Jan.) 1935.

toiditis caused by Friedländer's bacillus. Operation was performed in 5 of these cases, and in 2 the lesion healed spontaneously. All the patients recovered. He remarks that the course of the disease is not unlike that of a mucosus otitis, but the prognosis appears to be much better than that in infections caused by the type III pneumococcus. This observation is contradictory to what is found in the older literature, in which an infection with Friedländer's bacillus was regarded as one of great importance. Baum states that the pneumococcic otitis media found in children gives scant subjective symptoms. In these the outstanding complaint is of fulness in the ear. On otoscopic examination the drum membrane appears only slightly red and there is little or no bulging. The pus, however, will yield the same type of pneumococcus as is found in the sputum.

Goldman, Schwartzman and Herschberger [11] discuss the possibility of prophylactic vaccination against intracranial complications following mastoiditis caused by the type III pneumococcus. Between 1931 and 1934 25 patients were given 6 intradermal injections of a vaccine, the first being administered prior to operation and being given with a stock vaccine. The later injections were from autogenous vaccines. Of these 25 patients, 1 acquired meningitis five months postoperatively, whereas the other 24 recovered without any intracranial complications. The mortality, which had been 22 per cent for the four previous years, was found to drop to only 4 per cent, which is highly significant.

An unusual case is reported by Vener and Bower.[12] A child acquired acute purulent otitis media with a spontaneous perforation of the membrana tympani. Within a short time tetanus set in, and death occurred. The pus from the ear yielded tetanus bacillus in a pure culture, and when injected into an animal it reproduced the symptoms of tetanus.

PURULENT OTITIS IN THE EXANTHEMAS

Williams [13] in a study of 29,298 cases of scarlet fever found that 10 per cent of the patients showed aural complications; one third of these were children. Only 455, however, required a simple mastoidectomy. He recommends early paracentesis in all instances of purulent otitis which develops in the course of scarlet fever, arguing that the paracentesis is to be regarded as a preventive against further extension.

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11. Goldman, J. L.; Schwartzman, G., and Herschberger, C.: Prophylactic Vaccination Against Intracranial Complications Following Pneumococcus Type III Mastoiditis, Arch. Otolaryng. **21**:154 (Feb.) 1935.

12. Vener, H. I., and Bower, A. G.: Otogenic Tetanus: Report of a Case, J. A. M. A. **103**:480 (Aug. 18) 1934.

13. Williams, H. J.: Review of the Aural and Orbital Complications of Scarlet Fever at the Philadelphia Hospital for Contagious Diseases from 1922 to 1934, Ann. Otol., Rhin. & Laryng. **44**:110, 1935.

In connection with this, it is my experience that two types of infection result in the spaces of the middle ear during the course of scarlet fever. Infection which occurs within the first few days of the disease is usually of the type which has been described in the literature as acute necrotic otitis. This is the type which tends to remain throughout life as chronic purulent otitis media, and, owing either to caries of the bone or to the fact that nature has attempted to heal the lesion, it results in the formation of a cholesteatoma. The otitic infections, on the other hand, which occur later in the course of scarlet fever are more likely to run the course which an ordinary infection with Str. haemolyticus usually takes. It does not seem clear to me, therefore, just what purpose is accomplished by an early paracentesis performed as a preventive of complications, because it is an established fact that the development of coalescent mastoiditis is dependent on the resistance afforded by the protective forces of the body and on the local necrosis which causes bony cell walls to coalesce owing to continued pressure on nutrient blood vessels and to other factors well known in the mechanics of destruction of pneumatized bone cells, rather than on the establishment of drainage. By no stretch of the imagination could a myringotomy obviate the production of acute necrotic otitis. Nor do I know how the drainage could stop the factors referred to, which play their essential rôle in producing mastoiditis requiring surgical intervention.

A case in point is reported by Lifschitz.[14] In a girl 1½ years old symptoms of meningeal involvement made their appearance during the course of scarlet fever, which was further complicated by diphtheria. Bilateral otitis media developed, with postauricular swelling on the right side. At operation a sequestrum was found involving the entire right temporal bone. In this instance death resulted from hemorrhage from the internal carotid artery.

Sato [15] reports the presence of acute purulent otitis media in 14 of 75 cases of diphtheria of the upper respiratory tract. In only 5 of these, however, was the diphtheria bacillus isolated. In the majority of instances no characteristic clinical picture presented itself, and the significant pyogenic membrane was rarely found. This report does not accord with the findings of most other observers, and his countryman Nakashima [16] reports a case in which the culture from the pus in the middle ear yielded the diphtheria bacillus in pure culture; in this case the membrane was also present.

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14. Lifschitz, B. M.: A Case of Sequestration of the Temporal Bone in Scarlatinal Mastoiditis, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **24**:363, 1935.

15. Sato, Y.: Ueber Diphtherie des Mittelohres, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **24**:30, 1935.

16. Nakashima, T.: Diphtheria of the Middle Ear, Otologia, Fukuoka **8**:110, 1935.

MALNUTRITION AND INFECTIONS OF THE MIDDLE EAR IN INFANTS

Paschlau [17] feels that when no other cause is found for the inanition and diarrhea in infancy an antrotomy is indicated. This is borne out by the statements of ten Bokkel Huinink.[18] Ottoni de Rezende [19] takes the same view. The latter feels that because of the patent eustachian tube in the infant pressure pain is absent. Furthermore, the difficulty of properly viewing the membrana tympani owing to its horizontal position at this age renders accurate diagnosis of an infection of the middle ear almost impossible. Litschkus [20] is not so drastic in his recommendations. He takes the view that since acute purulent otitis media takes place frequently in infancy and since this condition is known to be the cause of intestinal intoxication, a paracentesis should be performed in all instances, since proper treatment of an otitic infection will cause the diarrhea to cease. I cannot, however, agree with his statement that an unnecessary myringotomy does no harm. All otolaryngologists see unusually large numbers of children whose hearing is faulty and whose parents usually attribute the defect to no other cause than one or more attacks of acute purulent otitis, with the inevitable repeated paracentesis. Even so simple a procedure should not be passed over lightly as a totally harmless one. The tympanic membrane does play a part in the transmission of sound waves. Any disruption of its intactness unquestionably must cause some impairment to the function of hearing. Of far more importance in this connection is the work of Bilinkis and Neiburg.[21] Of 25 patients who came to autopsy and the cause of whose death was termed malnutrition, the middle ear showed a pathologic picture in all. These authors feel that the type of otitic infection which they found is not of hematogenous origin but is rather the primary focus which produces general toxemia in infancy. They were unable to account for the symptomless course of this type of purulent otitis.

When one considers this question in its broader implications, one is impressed by two things. In the first place, it would seem, according to the view of some writers, that in all instances asthenia and dehydration are the direct result of otitic infection and can be expected to

17. Paschlau, G.: Mittelohr und Warzenfortsatzentzündungen im Krankheitsgeschehen des Säuglings und Kleinkindes, Kinderärztl. Praxis **6**:218, 1935.

18. ten Bokkel Huinink, A.: Otitis Media in Sucklings, Maandschr. u. kindergeneesk., **4**:219, 1935.

19. Ottoni de Rezende, M.: The Acute Middle Ear Infection and Acute Mastoiditis in Sucklings and Their Relation to Disease of Children, Rev. oto-laring. de São Paulo **2**:377, 1934.

20. Litschkus, M.: Die Otitiden der Säuglinge und der Zusammenhang zwischen den Otitiden und den Intoxicationen, München. med. Wchnschr. **81**:1492, 1934.

21. Bilinkis, G. S., and Neiburg, J. E.: Histopathology of Latent Otitis in Sucking Infants, Sovet. pediat., no. 2, 1934, p. 14.

clear up once the purulent condition has been eliminated. In the second place, one can view the situation as presenting a problem yet to be solved. Surely the ear does not act as the sole source of a generalized infection. Cullom [22] states that since 85 per cent of all otitic infections are induced by primary suppuration in the nasopharynx or in the sinuses, it is more logical to consider sinusitis as the inciting factor in the diarrheas of infancy. This view seems logical. It also adds to the belief that the operations on the ears of infants to alleviate intestinal intoxication are based on empirical rather than on scientific data. Finally, much of the work of the French authors on osteomyelitis of the temporal bones of infants has to be integrated with the other studies that have been made of infantile diarrhea and dehydration. This whole question remains to be reevaluated and correlated.

OTITIS AND NEPHRITIS

The occurrence of acute hemorrhagic nephritis as an accompanying feature or as a complication of an acute infection of the middle ear presents nothing new. Statistical reports, however, have thus far been few. It is interesting, therefore, that Kindler [23] in a study of 2,500 cases of purulent otitis media found that in 2 per cent of those in which the condition was acute renal complications were present and that in 0.2 per cent the otitis occurred with chronic otorrhea. Löwy [24] found that 0.7 per cent of acute purulent otitis infections were associated with hemorrhagic nephritis. This happens mostly among children. These authors feel that acute hemorrhagic nephritis in the presence of an infection of the ear may be considered as an indication for operative intervention. In this contention they are borne out by Wolff.[25] The prognosis in general is good, and the nephritis usually will clear up after adequate surgical therapy has been instituted. My own experience accords with this.

A rare complication of acute otomastoiditis is reported by Petterino-Patriarca,[26] who describes a case in which acute otitis media was followed by the Brown-Séquard syndrome. This consists of mydriasis and widening of the palpebral fissure. Only 3 other cases have been reported in the literature. After mastoidectomy the syndrome disap-

22. Cullom, M. M.: The Association of Sinus Disease and Middle Ear Infection, J. A. M. A. **103**:1695 (Dec. 1) 1934.

23. Kindler, W.: Die Beziehung zwischen Mittelohrentzündung und Nierenerkrankung, Med. Welt **8**:1761, 1934.

24. Löwy, K.: Akute Mastoiditis und Nephritis, Monatschr. f. Ohrenh. **69**: 690, 1935.

25. Wolff, S.: Beitrag zur Frage der Beziehung zwischen Mittelohrentzündung und Nierenerkrankung, Med. Welt **9**:161, 1935.

26. Petterino-Patriarca, A.: Sindrome oculare di Brown-Séquard causata da otomastoidite acuta, Riv. oto-neuro-oftal. **12**:284, 1935.

peared within nine days. The author feels that the development of this unusual syndrome was due to the fact that the otitic infection gained access to the pericarotid sympathetic plexus through a dehiscence in the carotid canal.

CHRONIC PURULENT OTITIS MEDIA

Wirth [27] agrees with Voss, Lange and Hesse that Bacillus proteus and allied gram-negative bacilli play an important rôle in the production of chronic infections. Despite this, however, he feels that the rôle which is exerted by transient infections of the middle ear in infancy is far more important in the development of chronicity. Infantile tuberculosis of the middle ear, usually of the bovine type, will produce a chronic purulent condition, but hyperplastic otitis and disturbances of pneumatization must be held responsible in most instances for the chronic purulent otitis which occurs in later life. This view is now generally held. It is the consensus among otologists that chronic otitis generally can be traced to early disturbances of pneumatization in infancy.

Simonetta [28] conducted histologic studies on aural polyps. He divides them into four types—the granulomatous, the plasmoma, the pseudomucoid and the one which shows giant cells. He states that all of them are inflammatory in origin. This accords with the well known view of Deuch.

An article which must be read in the original to be appreciated is that of Hoshiya,[29] whose experimental work on the formation of secondary otitic cholesteatoma is portrayed in fifty-two excellent photomicrographs. His studies were conducted on 700 mice and 20 guinea-pigs. His work proves that a secondary cholesteatoma is the direct result of an ingrowth of squamous epidermis. In order for this tumor to develop, it is necessary that either a marginal perforation or one located in the attic region be present. Only when the malleus is bound down by adhesions to the inner wall is it possible for a cholesteatoma to form in the presence of a central perforation. This work on animals bears out the findings of Wittmaack and of Almour [30] in the study of this condition in human subjects. The contention of Lange and Berberich that epithelioma grows into the middle ear through an intact drum was

27. Wirth, E.: Experimentelle Untersuchung zur Bakteriologie und Pathologie der chronischen Mittelohrentzündungen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **37**:316, 1935.

28. Simonetta, B.: I polipi dell'orecchio medio: Considerazioni sulla lora struttura (con speciale reguardo alla questione dei microcolesteatomi), e sui loro rapporti con gli pseudo-botriomicomi, Oto-rino-laring. ital. **4**:535, 1934.

29. Hoshiya, H.: Experimental Studies Concerning the Formation of Secondary Otitic Cholesteatoma, Mitt. d. med. Akad. zu Kioto **13**:1295, 1935.

30. Almour, R.: The Significance of the Squamous Epithelium in the Cause and Repair of Chronic Middle Ear Disease, Tr. Am. Acad. Ophth. **35**:357, 1930.

not substantiated by the work of Hoshiya. In view of this work it seems that otologists should cease wasting their energy in attempting to attribute a cholesteatoma to a disturbance of the cholesterol metabolism. Drennowa [31] studied the cholesterol content of the blood in cases of cholesteatoma with the view of finding a diagnostic test for the recognition of this condition. He found that the general cholesterol metabolism had no bearing on the presence or absence of cholesteatoma, but he feels that there must be a local disturbance of the cholesterol metabolism within the middle ear, since in several cases of chronic purulent otitis media with granulation and polyps a large number of cells were found which took the Sudan stain.

Secondary cholesteatoma was unquestionably the result of an ingrowth of epidermis into the tympanic cavity from the external canal. Whether or not a tumor develops depends solely on the type of anatomic structure over which the epidermis attempts to grow. It is distinctly a reparative process and should be viewed as such. I cannot consider seriously and further discuss articles which erroneously attempt to label secondary cholesteatoma as a constitutional disease. To do so is to negate accepted fundamentals in pathology.

An unusual case of primary cholesteatoma is reported by Beck.[32] A patient 20 years of age had deafness and facial paralysis. The middle ear was normal to otoscopic examination. A roentgenogram showed destruction of the petrosal tip. At operation both the middle ear and the mastoid process were found to be normal, but the labyrinth was opened and the dura was elevated in the area of the middle cranial fossa and a large cholesteatoma was found occupying the entire pyramidal apex. Recovery followed the operative removal of the lesion.

THERAPY

The value of physical therapy in otologic practice is finally beginning to take its proper place. One has but to look back a few years to find glowing reports of the efficacy of physical agents filling the literature. I feel that a great deal of the enthusiasm can be attributed to the manufacturers of ultraviolet ray machines, diathermy apparatus and other devices, whose detail men "pepped up" the unsuspecting practitioner. After mature judgment of the status of physical therapy otologists are now in a position really to estimate its worth.

Hollender [33] in a well written paper states that physical therapy is to be considered only as an aid and not as a permanent cure. Zinc

31. Drennowa, K. A.: Cholesteatoma und Cholesterin des Blutes, Ztschr. f. Hals-, Nasen- u. Ohrenh. **37**:212, 1935.

32. Beck, K.: Ueber ein Fall von primären Cholesteatom an der Felsenbeinspitze, Ztschr. f. Hals-, Nasen- u. Ohrenh. **37**:117, 1935.

33. Hollender, A. R.: Scientific Status of Physical Therapy in Otology. Laryngoscope **45**:471, 1935.

ionization has its place. Heat and the infra-red rays are of value in the relief of pain associated with otitic infection but do not stop progress of the lesion. The ultraviolet rays have distinct value in the treatment of tuberculosis of the middle ear. Regarding zinc ionization, Morison [34] states that it is of value only when the infection is limited to the middle ear and that it is worthless if necrosis of the bone or cholesteatoma is present. The treatment of otitic tuberculosis with ultraviolet rays is considered by Weinstein and Bendove,[35] but no general rules are laid down. Each patient must be treated individually. The real tuberculous otitis is benefited only when the ultraviolet rays are applied directly, and the treatment should be given at intervals of from two to five days. Of their 12 cases, they had a favorable result in 10.

ACUITY OF HEARING AFTER RADICAL MASTOIDECTOMY

Many patients with chronic purulent otitis media of the so-called dangerous type, which possesses the inherent potentiality of producing a fatal intracranial lesion, are carried along, and operation too is often delayed because it is feared, either by the patient or by his physician, that loss of practical hearing will follow radical mastoidectomy. It is significant in this connection to cite the findings of Alföldy,[36] who reports on 65 cases in which radical mastoidectomy was done and 1 in which a so-called conservative radical operation was performed. In 83.43 per cent the hearing after operation was decidedly improved. In 10.66 per cent there was no change in the status of hearing. Only 6 per cent of the patients were observed to show a distinct loss in their acuity of hearing over that which existed prior to the surgical procedure. He also quotes statistics given by Björkmann, who in a series of 55 radical mastoidectomies noted an improvement in the hearing of 32 per cent of his patients, an unchanged status in 40 per cent and a reduction of hearing of the remaining 28 per cent.

This is in accord with my own observations. During the years of my practice I have seen many patients whom I have advised to have a radical mastoidectomy performed for the cure of a lesion which I consider a potentially fatal disease but who disregarded my advice, either on their own account or because their personal physician dissuaded them. I could not assure them that their hearing would be no worse after the operation. These patients later presented themselves either to me or to my colleagues with intracranial complications that had in many

34. Morison, G. P.: Chronic Otorrhea, with Special Reference to Zinc Ionization and Ultraviolet, Arch. Phys. Therapy **16**:101, 1935.

35. Weinstein, A. J., and Bendove, R. A.: Ultraviolet Treatment in Chronic Otitis Media in the Tuberculous, Arch. Phys. Therapy **15**:419, 1934.

36. Alföldy, E.: Die Hörfähigkeit nach radikalen Ohroperationen, Acta otolaryng. **21**:368, 1934.

instances extended beyond the stage at which surgical intervention could afford them relief. It is my contention that the preservation of life is of far more importance than the preservation of hearing. The report of Alföldy affords evidence that the delay in operating is unnecessary and, in view of the consequences, unwise. Furthermore, I have now followed several patients through years of observation, and have found that at the end of ten years the patient who had no operation invariably shows a distinct loss of acuity of hearing in any event. On the other hand, in my cases in which operation had been undertaken, the loss in hearing in most instances was substantially reduced. Thus if 2 patients are seen at about the same time and tested for loss of hearing, and if one is operated on and the other is carried along under continuous local treatment, reexamination of both at periods of from five to ten years will reveal that the patient who was operated on and whose lesion was cured will show the better acuity of hearing in the majority of instances. In addition, this patient will have escaped the continued threat of an intracranial complication.

SINUS THROMBOSIS

Hofer [37] distinguishes the various types of metastases which follow septic sinus thrombosis in children. The types which he considers are the cranial, thoracic, abdominal and skeletal. He notes that in childhood metastatic lesions of the bone, joints and muscles are rare. At this stage of life the secondary lesions are likely to attack the more vital structures and to result in more serious complications.

Since septic sinus thrombosis is most frequently the result of an infection caused by Str. haemolyticus, it is interesting to note the reports of Wolf [38] and of Smith and Miller.[39] The former reports a case of septicemia caused by Streptococcus albus secondary to sinus thrombosis in a woman 40 years old. The blood culture was positive for this organism. At operation a mural thrombus was found. The latter authors report a case of pyemia subsequent to thrombosis of the sigmoid sinus which developed in the course of chronic purulent otitis media in which the blood culture yielded B. proteus. This observation is in accord with the work of Wirth, who feels that B. proteus plays an important rôle in this disease.

It is not unusual to find complete obliteration of the lateral sinus by a thrombus which has become organized. This phenomenon is the

37. Hofer, L.: Ueber Metastasenbildung bei otogener Pyämie im Kindesalter, Monatschr. f. Ohrenh. **68**:713, 1934.

38. Wolf, G. D.: Staphylococcus Albus Septicemia Secondary to Mastoiditis and Sinus Thrombosis: Operation and Recovery; Report of Case, Laryngoscope **45**:227, 1935.

39. Smith, A. B. P., and Miller, S.: Lateral Sinus Thrombosis Due to Bacillus Proteus, Lancet **1**:862, 1935.

end-result of nature's effort to effect the healing of a suppurative focus within the blood vessels. Simeoni [40] reports such a case. A woman 24 years of age presented a completely thrombosed sinus at operation. At no time during the course of her disease were there any signs or symptoms of a septic sinus thrombosis. While this is not unusual, I question the contention of the author that this was brought about because she had been treated prior to operation with injections of polyvalent vaccine. I personally have seen no good result from the use of vaccine therapy in any type of severe otitic infection. Perhaps the perfection and the advance in bacteriologic research within recent years may afford a real basis on which otologists may more intelligently employ this type of therapy. Certainly, the use of vaccines by Schwartzmann and his confreres in the prevention of intracranial complications when the type III pneumococcus is producing the otitis would seem to open a field for a renewed study of this topic and perhaps for a better therapeutic result than has heretofore been obtainable. As yet the question remains open, and one observation must not be made the basis for a generalization.

Primary thrombosis of the jugular bulb occurs rarely in adults. Dintenfass [41] reports such a case in which the mastoid process at operation was found to be normal. The lateral sinus also appeared normal, but when it was opened fluid pus and a thrombus were found in the bulb. Str. haemolyticus was the causative agent. While the blood culture was negative, the clinical picture was typical of sepsis. Again it is timely to call attention to the fact that the blood will contain organisms which will grow on culture only when the protective forces of the body have been unable to localize the infection at its point of origin within the wall of the vein. A thrombus is essentially a protective mechanism. It is formed to prevent the access of bacteria into the general circulation. Only when this protective thrombus becomes disintegrated by the organisms does the blood stream become invaded. Consequently, the clinician must guide himself much more by the clinical picture presented by his patient than by what the laboratory can tell him. A septic thrombus can be present within the lateral sinus, although the blood culture is negative. To wait until a positive blood culture is obtained is to invite unnecessary delay in the removal of a focus of infection which subsequently may prove fatal.

From a study of 65 cases of sinus thrombosis following acute suppurative lesions of the tympanomastoid area and 139 cases in which

40. Simeoni, C.: Tromboflebite asintomatica del seno laterale di origine otitica. Considerazioni sulla vaccinoterapia, Arch. ital. di otol. **45**:783, 1934.

41. Dintenfass, H.: Primary Jugular Bulb Thrombosis in an Adult, Arch. Otolaryng. **20**:422 (Sept.) 1934.

chronic purulent otitis media was the inciting factor, Frühwald [42] concludes that the signs of sepsis make their appearance postoperatively much sooner when a chronic lesion is the underlying factor. He noted that the ligation of the internal jugular vein is no guarantee of the prevention of metastatic lesions. The latter observation is not new. The ligation of the internal jugular vein does not localize the lesion entirely, because the petrosal veins, the condyles and the vertebral veins are not shut off and are potentially avenues through which infection can be carried into the general circulation. These, of course, constitute only the more important channels which afford access to the blood stream.

Treatment.—Feuz [43] discusses the views held by the opposing schools anent the value of operation on the sigmoid sinus and the jugular vein in cases of septic sinus thrombosis. He feels that those who hesitate to operate may spare an occasional patient an operation, but only because this patient in reality did not actually need one. He contends that in the majority of instances it is dangerous to the patient not to be operated on. In arriving at this conclusion he carefully considers the fact that spontaneous healing of sinus thrombosis may occur.

Wenner and Cone [44] consider the use of intramuscular injection of an extract of the adrenal cortex in cases of pyogenic infection of otitic origin. In the instances in which they used this type of medication the temperature dropped, the general condition improved and the blood picture reflected this improvement. In addition, the postoperative course was much less stormy. They feel that in all probability the action of this is due to the stimulation of the leukocytes and to the increase in their phagocytic activity.

Boller and Feuz [45] claim that the therapeutic effect of blood transfusion in cases of sepsis can be much increased by the injection of 0.75 mg. of epinephrine hydrochloride five hours after the transfusion has been given.

Specific therapy in treatment of streptococcic infection of the blood stream following otitis has been tried but has not yet proved very effective. Baum [46] presents a medium for the specific treatment in cer-

42. Frühwald, H. E.: Beitrag zur Pathologie und Klinik der otogenen Sinusphlebitis, Acta oto-laryng. **21**:394, 1935.

43. Feuz, J.: Remarques sur le traitement des septicemies d'origine otique, Rev. de laryng. **55**:969, 1934.

44. Wenner, W. F., and Cone, A. J.: Use of Extract of the Suprarenal Cortex in Pyogenic Infections, Arch. Otolaryng. **20**:178 (Aug.) 1934.

45. Boller, R., and Feuz, E.: Ueber die Steigerung der therapeutischen Wirkung der Bluttransfusion bei Sepsis, Wien. Arch. f. inn. Med. **27**:23, 1935.

46. Baum, H. L.: A Method of Specific Treatment in Certain Streptococcus Infections, Arch. Otolaryng. **20**:504 (Oct.) 1934.

tain types of streptococcic infection. A culture should be made of immune serum, which can be obtained from persons convalescing from scarlet fever and other streptococcic infections. When a patient with a general streptococcic sepsis presents himself, a culture should be made from the focus either in the blood or in the spinal fluid, and this should be tested for agglutination against the serums which have been obtained previously. The agglutinating power is then determined for the serums against the specific streptococcus obtained from the patient. Not all streptococci will be agglutinated by the same serum. The potent serum should be administered intramuscularly or, if necessary, intravenously to the patient with the septic condition. He advises that a large dose should be given at the first injection.

THE PETROUS PYRAMID

Anatomic Data.—Many observations have been made concerning the structure of the perilabyrinth. The perilabyrinth at birth is composed of diploic spaces, which are filled with either red or fatty marrow. During the process of pneumatization the perilabyrinth may become pneumatic owing to the influence of the lining membrane of the tympanic cavity. On the other hand, it may remain diploic throughout life. When a severe disturbance in the process of pneumatization has occurred, sclerosis of this part of the temporal bone may result. It is known that the perilabyrinthine space does not necessarily correspond to the structure of the mastoid process as far as the distribution of air-containing cells is concerned. Myerson, Rubin and Gilbert [47] make this clear in their study of 200 specimens of the petrous portion of the temporal bone. The routes by which pneumatization can occur are four. Tobeck [48] describes them as: (1) the posterior one, in which the pneumatic cells stretch from the antrum around the posterior canal along the upper part of the posterior surface of the internal auditory meatus to the tip, (2) a superior one, in which the pneumatization occurs from the epitympanic recess above the semicircular canal underneath the superior petrosal surface to the tip, (3) an inferior one, from the hypotympanum underneath the labyrinth and the internal auditory meatus to reach the apex, and (4) an anterior route, which extends from the eustachian tube above the carotid artery to the tip. Ziegelman [49] studied

47. Myerson, M. C.; Rubin, H. W., and Gilbret, J. G.: Report on the Anatomical Study of Two Hundred Petrous Portions of the Temporal Bone, Laryngoscope **45**:159, 1935.

48. Tobeck, A.: Anatomische Untersuchungen über die Pneumatisation von Felsenbein und die Wegleitung zur Spitze, Ztschr. f. Hals-, Nasen- u. Ohrenh. **37**:152, 1935; Ueber Pyramidenzellen und Pyramidenzelleneiterungen, Zentralbl. f. Ohrenh. **40**:193, 1935.

49. Ziegelman, E. F.: The Cellular Character of One Hundred Temporal Bones: Clinical and Surgical Significance, Ann. Otol., Rhin. & Laryng. **44**:3, 1935.

the cellular character of 100 temporal bones, and his findings coincide with those of Zuckerkandl. In 20 per cent of his cases pneumatization was present in various portions of the petrous pyramid. Myerson, Rubin and Gilbert found 11 per cent of their specimens to be pneumatized.

Wilson,[50] who has examined many temporal bones of infants, still contends that the entire perilabyrinth is diploic in all instances. From his studies he has never been able to demonstrate any air-containing cells. The specimens which were shown by Guild[51] definitely refute this, because they show air-containing spaces within the perilabyrinth, the bony walls of which are lined by a mucoperiosteum, which is identical with that found in the pneumatic mastoid process. From a practical standpoint, it makes little difference whether a petrous pyramid exclusive of the otic capsule is partially or wholly pneumatized. When a suppurative process is present in this vicinity, it requires the same type of surgical treatment that a suppurative focus anywhere else in the body does; namely, adequate drainage must be established. It is only when a petrous pyramid is entirely diploic that a different type of pathologic process occurs. Osteomyelitis develops, but this occurs rarely except in infants and young children. From a practical standpoint the clinician can still guide himself as to the character of the petrous pyramid by the extent of pneumatization present in the mastoid process. In my experience no instance of petrositis has developed in which at operation a sclerotic or a diploic mastoid process was found. In addition, a roentgenologic examination will soon reveal the character of the petrous pyramid. Thus, it becomes possible to differentiate between what, after all, is an academic question and what is of practical import to the clinician. While the scientific discussion regarding details proceeds, the clinician is confronted with a practical bedside problem which he must solve if he is to institute adequate therapy. It is here that the clinical picture and roentgenograms help him toward a solution.

Pathologic Picture.—The nomenclature of petrositis is still indefinite and too vague, and what constitutes acute petrositis and what chronic infection remain a question for solution. Jones[52] states that chronic infections are the most serious, and he feels that these are the result of acute infection superimposed on a prolonged process. He contended that the entrance of a new organism into a previously formed cavity activates a process already present and at the same time produces a situation in which free drainage of pus becomes impossible because

50. Wilson, J. G.: Normal and Pathological Anatomy of the Petrous Pyramid, Ann. Otol., Rhin. & Laryng. **44**:1050, 1935.

51. Guild, S. R.: Normal and Pathological Anatomy of the Petrous Pyramid, Ann. Otol., Rhin. & Laryng. **44**:1013, 1935.

52. Jones, M. F.: Normal and Pathological Anatomy of the Petrous Pyramid, Ann. Otol., Rhin. & Laryng. **44**:1038, 1935.

the original pathway may have become obstructed by the swelling of its membrane, by detritus or by adhesions, and that thus the lesion is produced.

I cannot view chronicity in petrositis in any different light from that in which I view chronicity in the other otitic infections. The element of time should not be considered. Chronic petrositis is a purely pathologic process, which differs in no way from the chronic otorrhea which is the result of destruction of bone elsewhere in the temporal bone. In chronic petrositis the otologist is confronted by a lesion which occurs within the perilabyrinth and which has established for itself adequate drainage through the formation of a fistula, so that no symptoms of retention of pus present themselves. Nevertheless, continuous destruction of bone occurs, and the pus formed finds its way out easily into the middle ear and the external canal, through an incision in the mastoid or through variously located fistulas and produces a chronic purulency. In the specimens I have studied slight local changes shown in microscopic sections in either definitely located or widely separated areas are not significant of chronic petrositis. Similar microscopic pictures in specimens from the tympanomastoid area would also not have significance. I cannot ascribe to them in the petrosa characteristics not allocated to them elsewhere. I cannot ascribe to these small local tissue reactions sufficient significance to make these findings characteristic of a chronic lesion of the bone.

Clinical Data.—Glauberman [53] reports 3 cases of petrositis in which the patient recovered after operation. In 2 instances a simple mastoidectomy was sufficient to uncover the lesion. Zanni [54] cites a case in which the condition was cured by the Voss epitympanic operation. He believes that therapy with autogenous vaccine may have helped in the cure in his case. Hall [55] also reports a case in which recovery followed a simple operation on the mastoid. The reason for the report of this case is a plea for conservatism in the treatment of petrosal empyema. This question will be discussed later. Almour and I [56] report 10 additional cases. Nine of the patients presented fistulas. Nine recovered and 1 died from an abscess of the brain which subsequently developed. We stress the fact that there is no one method of operation which is suitable for all patients with petrosal suppuration. Nor does every patient require surgical therapy. We make a plea for differentiation of

53. Glauberman, S.: Concerning Abducens Palsy of Otitic Origin, Sovet. vestnik oftal. **5**:311, 1934.

54. Zanni, G.: Un caso di sindrome di Gradenigo da osteite della rocca petrosa, Oto-rino-laring. ital. **5**:137, 1935.

55. Hall, C.: Conservatism in Petrosal Empyema, Laryngoscope **45**:421, 1935.

56. Kopetzky, S. J., and Almour, R.: Report on Ten Cases of Suppuration in the Petrosal Pyramid, Ann. Otol., Rhin. & Laryng. **44**:59, 1935.

cases and for a specific selection of the therapy to fit the condition presented in the given case. Tsuda [57] cites the autopsy findings in a case in which fatal meningitis developed subsequent to a petrosal suppuration. At autopsy all the perilabyrinthine cells were found to be infected, and the infection extended to the apex, where a rupture had occurred.

Gradenigo's Syndrome.—Despite what has been written in recent years concerning the confusion which the term Gradenigo's syndrome has caused in the evaluation of suppurative petrositis as a clinical entity, this phrase still appears in the literature. If otologists would but comprehend that the Gradenigo syndrome is not synonymous with suppurative petrositis, much would be accomplished in the understanding of a disease which always carries a threat to life. This syndrome is a triad. It is present when otorrhea, palsy of the abducens nerve and neuralgia of the trigeminal nerve are present at the same time. This syndrome does not necessarily indicate the presence of a suppurative focus within the petrous pyramid. It is far better, and it will serve a more useful purpose if suppurative petrositis is classified into a type which presents palsy of the abducens nerve and one which does not have this sign. Paralysis of the abducens nerve is not necessarily an accompanying factor of petrositis. I have held that the factors produced by suppurative petrositis may be entirely intrapetrosal or peripetrosal in origin. The Gradenigo syndrome is of peripetrosal origin, appears late in the course of the disease and is a meningeal reaction.

To emphasize this, Rotenberg and Truschin [58] comment on the fact that Gradenigo's syndrome can be caused by a toxic focus which is located extradurally. They observe that an intradural focus or a fulminating meningitis can also produce this syndrome. Petrositis can also give rise to the Gradenigo syndrome. As far as therapy is concerned, these authors feel that in an acute stage operative intervention in petrositis is contraindicated, an opinion with which I am not in accord. Mitchell and Silverstein [59] report a case in which Gradenigo's syndrome followed a simple mastoidectomy. At autopsy the cause for the condition was found to be purulent bacillary meningitis and an abscess in the fourth ventricle. Eves [60] cautions against considering

57. Tsuda, T.: Contributions to the Relationship of Purulent Meningitis to Petrositis, Otologia, Fukuoka **18**:901, 1935.

58. Rotenberg, J. G., and Truschin, A. W.: Concerning Gradenigo's syndrome, Sovet. vestnik otol. **27**:315, 1934.

59. Mitchell, E. K., and Silverstein, A.: Gradenigo Syndrome and Suppuration of the Cerebral Ventricles as Complications of Acute Suppurative Otitis Media, Laryngoscope **45**:214, 1935.

60. Eves, C.: Diagnosis of Acute Suppuration of the Petrous Pyramid, Ann. Otol., Rhin. & Laryng. **44**:97, 1935.

the Gradenigo syndrome as synonymous with petrositis. He evaluates the various diagnostic features and points toward orbital pain and otorrhea as being far more important than the presence of palsy of the abducens nerve. Oliva [61] also stresses the importance of retro-orbital pain and cites 2 cases in which palsy of the abducens nerve was associated with it.

The value of roentgenologic examination in the diagnosis of petrositis is stressed by Taylor [62] and by Kraus.[63] Taylor makes a fine differentiation between the finding of clouding and the evidence of destruction which can be completely visualized by the roentgen rays. He also stresses the importance of separating those cases in which the suppurative focus has adequate drainage from those in which drainage is either blocked or inadequate. Taylor states that the eventual determination of the type of therapy to be employed must rest with the clinician. Kraus cites 3 cases in which protracted otorrhea, unilateral headache and ocular pain led to a diagnosis of petrositis. This was confirmed by roentgenologic examination. He states that the location of the suppurative focus within the petrous pyramid should be the determining factor in the selection of the type of operation which is to be undertaken for its cure.

Friesner and Druss [64] made a study of autopsy material in 24 cases. They make the point that the lesions they found did not necessarily extend toward the apex of the pyramid in all cases. They incline to the belief that the posterosuperior route is the most frequent pathway of infection. With no clinical data or reported observations in this article, they conclude, among other things, that suppuration in the petrous pyramid tends to heal spontaneously. Subsequently, in another report, Friesner, Druss, Rosenwasser and Rosen [65] published their observations and the histories of 7 cases in which the lesion healed spontaneously. The data on spontaneously healed lesions of the petrous pyramid are too meager to form a basis for such a generalization, particularly when contrasted with these authors' own report of 24 autopsies of persons with this type of infection. Furthermore, all who are studying this lesion and its clinical manifestations have observed cases in which the lesions healed spontaneously and others in which they healed

61. Oliva, R.: Eye Symptoms in Diseases of the Petrous Tip, An. de otorino-laring. d. Uruguay **5**:69, 1935.

62. Taylor, H. K.: Suppuration in the Pneumatic Petrous Apex, Am. J. Roentgenol. **33**:767, 1935.

63. Kraus, L.: Röntgenologische und histologische Untersuchungen zur Petrositis, Ztschr. f. Laryng., Rhin., otol. **26**:209, 1935.

64. Friesner, I., and Druss, J. G.: The Fundamentals of Therapy, Ann. Otol., Rhin. & Laryng. **44**:1102, 1935.

65. Friesner, I.; Druss, J. G.; Rosenwasser, H., and Rosen, S.: Suppuration of the Petrous Pyramid, Arch. Otolaryng. **22**:659 (Dec.) 1935.

after the performance of a simple mastoidectomy. This too is not any criterion on which to base a generalization, for the physician at the bedside must make a decision as to whether or not the condition he is treating will heal spontaneously or whether he should await intracranial manifestations before he shall become more than a bedside observer of a developing endocranial lesion. I cannot agree with the statement "our belief is that suppuration confined within the petrous pyramid has a marked tendency toward spontaneous cure." On the basis of the 45 cases from my own practice in which after diagnosis, while the lesion was still intrapetrosal, the patient was submitted to surgical therapy and recovered, I hold the view of these observers as untenable. There are definite clinical manifestations which tend to show that a condition is progressing toward spontaneous recovery. It is hardly proper in a review of bibliographic material to digress to write a new paper on this one feature of the topic, but in the current published reports these data can be found. What is urgently needed now is an unequivocal statement of signs, symptoms and data from clinical study which will clearly differentiate the condition which is in recession from that which is rapidly progressing toward the inevitable intracranial lesion and death.

I am aware that there are those who continue observation and local topical treatment on recognized cholesteatoma of the spaces of the middle ear until intracranial complications threaten and the endocranium is invaded and that this same type of otologist probably would watch petrosal lesions also until the cranial cavity showed evidence of invasion. With this school of thought I am not in accord.

Furthermore, if one considers the report of Myerson, Rubin and Gilbert [66] on the frequency with which pathways and fistulas are found, the preponderance of occurrences of the posterosuperior route is controverted. Myerson and his colleagues found lesions as follows:

Anterior to superior canal	14
Posterior to superior canal	8
Posterosuperior border	3
Tubal	5
Peritubal	
Superior	5
Inferior	3
Not mentioned	5
Postero-inferior (jugular)	5
Arch of superior canal	4
Posterior or behind posterior canal	1

They also note the reports of multiple pathways and fistulas.

66. Myerson, M. C.; Rubin, H. W., and Gilbert, J. G.: Considerations on Suppuration of the Petrous Pyramid, Arch. Otolaryng. **22**:62 (July) 1935.

From this, as well as from the classic report of Ramadier,[67] it seems well established that the pathway of invasion will be located in other regions of the perilabyrinth more often than in the posterosuperior region. Almour's [68] observation that "the opinions of the various workers are separated only by the fact that most of them have seen and treated only one or at the most a few types of petrositis" is a shrewd summation of the divergence of views with which the current literature abounds. Nor is this to be wondered at in this early stage of the development of the general comprehension of this new clinical entity.

The perilabyrinthine space is the entire portion of the petrous pyramid which surrounds the otic capsule. All or any portion of it may be involved, and any disease in any portion is deemed petrositis. Consequently the suggestion of Guild,[51] Jones [52] and Almour [68] that the location of the lesion should be definitely indicated as existing in relationship with definitely known structures, such as the semicircular canal, cochlea, carotid artery or eustachian tube, not only will give a more descriptive designation to the lesion but will afford a far better indication as to the type of therapy required to reach it than does the generalization of the disease under the one term "petrositis." Ramadier has remarked that the diagnosis of the petrosal lesion should serve as an indicator of the type of therapy required for its cure. Eagleton [69] brings another point of view toward knowledge of this subject. He is a pioneer in this field, and his views must be given studied consideration. He states that the lesion rarely presents apical abscesses because the fatty marrow within the petrous perilabyrinth may be converted into red marrow, a tissue which is known to be resistant to infection. He attempts to present further evidence and states that in instances in which pneumatic spaces are present within the petrous perilabyrinth a threatened infection of these may cause a reversion of these air-containing chambers to their former state, when they will again contain bone marrow!

Since it has been shown that the lining membrane of the air-containing spaces within the petrous pyramid differs in no way from that of the mastoid cells, it is inconceivable that a return of red bone marrow could occur in one portion of the temporal bone which is threatened by infection and does not occur in the cellular structures in the mastoid process, which also in its original state, before the process

67. Ramadier, J.: L'ostéite profonde du rocher, Vie méd. **14**:931, 1933.

68. Almour, R.: Fundamentals of Therapy, Ann. Otol., Rhin. & Laryng. **44**: 1109, 1935.

69. Eagleton, W. P.: Meningitis: The Result of Disease of the Petrous Apex and Sphenoidal Base, Surg., Gynec. & Obst. **60**:586, 1935; The Fundamentals of Therapy, Ann. Otol., Rhin. & Laryng. **44**:1115, 1935.

of pneumatization set in, was a true diploic bone and contained bone marrow. Why should the air cells of the petrous pyramid be particularly favored? Why would it not be logical to conclude that similar structures, having undergone similar developmental changes and become fixed, should act in a similar manner when they become infected by the same causal agent? I cannot accept as a significant fact the disappearance of the air spaces in the pyramid, the destruction or absorption of its lining membrane and a replacement of bone marrow by migration in the face of bacterial invasion unless an entirely new pathologic picture in the bone is to be established. I believe that Eagleton observed bone marrow in this case, but I also believe that his deduction from his observations are not correct.

I wonder whether the specimen or specimens on which he bases his report may not have been histologically a plasmocytoma. Cappell and Mathers [70] report a case of a woman 57 years old, who had chronic purulent otitis on the right side for four years. Facial paralysis and palsy of the right abducens nerve made their appearance. An operation on the mastoid was performed, and the mastoid process was found filled with the tumor mass. The patient died; at autopsy the tumor was found to extend to the petrous apex, and on histologic examination it proved to be a plasmocytoma.

Complications of Petrositis.—Torrini [71] reports 6 cases of petrositis, in 3 of which a dissecting descending abscess complicated the clinical picture. In 1 case the pus found its way into the lateral pharyngeal wall. In another it made its way into the pterygomaxillary fossa, and in the third, into the suboccipital region. Takasaki and Shin [72] report an instance of acute mastoiditis on the left side caused by the type III pneumococcus, in which an abscess formed on the left side of the pharynx and then on the right side. Incision of both these caused a rapid cessation of the otorrhea. Sadowski [73] reports a case of a 68 year old diabetic patient, in whom at mastoidectomy an extensive involvement of the perilabyrinthine cells was found. After a protracted clinical course a lateral pharyngeal abscess developed, opening of which resulted in a quick recovery. In a 21 month old infant a retropharyngeal abscess developed following acute purulent otitis media. Boenninghaus [74]

70. Cappell, D. F., and Mathers, R. P.: Plasmocytoma of the Petrous Temporal Bone and Base of the Skull, J. Laryng. & Otol. **50**:340, 1935.

71. Torrini, L.: Contribute alla conoscenza della petrositis, Oto-rino-laring. ital. **5**:340, 1935.

72. Takasaki, T., and Shin, M.: A Cured Case of Otogenic Retropharyngeal Abscess, Otologia, Fukuoka **8**:536, 1935.

73. Sadowski, N.: Zur Kenntnis der otogenen Retropharyngealabscessen, Folia oto-laryng. orient. **2**:80, 1935.

74. Boenninghaus, G.: Ein Fall von otitischen Retropharyngealabscess im frühen Kindesalter, Arch. f. Ohrenh. **137**:360, 1933.

attributes these conditions to a dissecting abscess originating from the base of the skull in the region of the petrous bone. In a previous report by Almour and me two cases were cited of pharyngeal abscess which was the result of gravity drainage from suppurative petrositis. Seydell [75] also reported such findings (see also Aloin,[76] Bowers,[77] Eagleton, Grunert,[78] Muck,[79] and Myerson).

An unusual complication of a petrous suppuration was reported by Vigi and Baccarani.[80] In their case an abscess of the spleen was found at autopsy after their operation for this condition. However, I regard this abscess as a metastatic lesion. The pharyngeal abscesses described were in no sense metastatic lesions but were developed by rupture into contiguous tissues and under the influence of gravity burrowed their way downward in the deep muscle sheaths of the side of the neck, to erupt eventually on the posterolateral pharyngeal wall.

In the majority of cases in which meningitis followed acute suppurative petrositis the condition was the result of a break at the petrous apex or in the region of the internal auditory meatus. Kramm [81] presents histologic studies of 2 cases in which infection of the perilabyrinth traveled along the sublabyrinth cells to break into the aqueduct of the cochlea. When such a pathologic condition is present his advice is to open the cells from behind.

Therapy.—Jones [82] discusses the pathway of approach to the petrous pyramid. He presents microscopic evidence on which he bases his discussion for the various approaches toward suppuration in the petrous apex. Almour outlines a basis of surgical approach based on the clinical recognition of the location of the lesion within the petrous pyramid.

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75. Seydell, E. M.: Surgical Treatment of Suppuration of the Petrous Pyramid, Ann. Otol., Rhin. & Laryng. **42**:455, 1933.

76. Aloin, H.: Apropos des ostéites profondes de la pointe du rocher, Ann. d'oto-laryng. **3**:194, 1933.

77. Bowers, W. C.: Two Cases of Petrous Bone Abscess, Laryngoscope **38**: 412, 1928.

78. Grunert: Anatomische und klinische Beiträge zur Lehre von den intracraniellen Complicationen der Otitis, München. med. Wchnschr. **44**:1375, 1897.

79. Muck: Beiträge zur Kenntnis der otitischen Erkrankungen des Hirns, der Hirnhäute, und der Blutleiter, Ztschr. f. Ohrenh. **37**:174, 1900.

80. Vigi, F., and Baccarani, C.: Ascesso splenico come complicanza diuna petrosite purulenta acuta, Oto-rino-laring. ital. **5**:87, 1935.

81. Kramm, H.: Sublabyrinthäre Entzündung, Ztschr. f. Hals-, Nasen- u. Ohrenh. **37**:255, 1935.

82. Jones, M. F.: Pathways of Approach to the Petrous Pyramid, Ann. Otol., Rhin. & Laryng. **44**:458, 1935.

Abstracts from Current Literature

Ear

Acute Otitis Media. J. V. Cassady, J. Indiana M. A. **29**:17 (Jan.) 1936.

This article is an interpretation of the paper entitled "Otitis Media" by Prof. Georges Portmann, of the University of Bordeaux. Otitis media is classified into the simple and the necrotic. Simple otitis media is an inflammation of the mucosa without destruction of the epithelium; perforation is caused by pressure on the tympanum. The prognosis is excellent from the standpoint of life and of auditory function. Necrotic otitis media is a severe inflammation of the mucosa with destruction of the epithelium which always leaves sequelae. The perforation of the tympanic membrane is larger than occurs with the simple form. The destructive process heals by scarring and often leads to permanent auditory trouble.

Miller, South Bend, Ind. [Am. J. Dis. Child.]

Scientific Audiometry and Selective Application in the Design and Construction of Modern Deaf-Aids. A. Tumarkin, J. Laryng. & Otol. **50**:838 (Nov.) 1935.

Tumarkin shows that to provide an adequate hearing aid for a deaf patient—as the oculist and the optician together provide a visual aid—two things are necessary: accurate refraction of the hearing powers and an instrument designed in accordance with this refraction which will so magnify the ordinary sounds (e. g., of speech) as to compensate for the distortion of the auditory apparatus. The former objective can be achieved only by scientific audiometry, and the latter, only by selective amplification. The author points out that the range of pitch for speech extends from about 120 to 8,000 double vibrations. Normal hearing gives perception of from about 20 to 20,000 double vibrations. What happens when a patient is deprived of one end or the other of his hearing spectrum? Experiments show that while cutting out the lower range alters the timber of the spoken voice, it has little or no effect on its clarity or intelligibility. On the other hand, diminution of the upper range rapidly impairs intelligibility. The energy of speech sounds is to be found mainly in the lower pitches. Thus the fricative consonant th as in "thigh" lies high in the region of 6,000 double vibrations and represents only about one thousandth of the energy of the vowel a as in "awl," which lies at about 600 double vibrations. The higher notes must be produced with much more energy than the lower if the ear is to notice them; i. e., their threshold is much higher. The hearer cannot afford to lose his high tones, but he has ample tones to spare in the lower reaches. The phenomenon of masking is no recent discovery. It was first studied by Mayer in 1876 and has been extensively investigated by telephone laboratories. For our purpose the important fact is that, given a mixture of high and low tones, intensifying the low tones masks the high ones, but the converse is not true. The author further calls attention to the limitations of the older instruments and then gives his attention to the possibilities of scientific audiometry. He confines himself to air-conducted sounds. He reproduces a standard audiometer chart and shows the pitfalls that await the unwary in reading this chart. Then he shows that the liability of the limited point chart to misrepresent the true state of affairs could be obviated by a continuous band audiometer. This instrument usually incorporates two valves oscillating at radio-frequency. By modifying one or both rates of oscillation the valves can be made to heterodyne or beat at audio-frequency, and so one can obtain a pure note of any frequency—for instance, from 10 to 10,000 vibrations. This note can again be augmented or attenuated in decibels. If, then, it is suspected that

the patient has a resonance area at, for instance, 800 vibrations, the point can easily be settled. Such a machine is unfortunately expensive, and much research will have to be done before its use can be justified. If, however, the suggestion of a peak resonance in diseased ears should prove to be accurate, some instrument of this kind would be essential to discover it. If the aurist is able to state accurately, by means of his audiometer, which regions of his patient's auditory spectrum are affected most, and in particular whether an area of peak reasonance exists, the problem remains whether the manufacturer can produce a special instrument to suit the individual need. Admittedly, at present this is far from being realized, but the author believes that there are no insuperable obstacles, and he states that some of the ideas embodied in this article have already been put in practice. In the meantime, it is clear that the simple pathologic subdivision of deafness into the conductive and the perceptive type is inadequate. Patients must be considered in the light of the results of their audiometric tests if they are to be given the full benefits of the miraculous advances of modern science. He shows further the advances that have been made in the construction of microphones and telephones and suggests that the construction of a miniature moving coil telephone will mean a considerable advance in hearing aids. He also suggests a standard of sound intensity. He thinks that if resonance in diseased ears is found to be important, the principle of the band pass filter will be used more widely. Finally he suggests a filter system that cuts out certain frequencies, while it reduces intelligibility only slightly.

EDITOR'S ABSTRACT.

A METHOD FOR TESTING PATENCY OF THE EUSTACHIAN TUBE. F. ZÖLLNER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **140**:137, 1935.

Zöllner determines patency of the eustachian tube by producing excessive pressure in the nasopharynx. By this method he has determined that the eustachian tube is patent in all persons without disease of the ear. With pharyngeal catarrh patency is often reduced, although auditory disturbances may be absent. With exudation from the middle ear patency is sometimes normal, often slightly impaired and rarely nonexistant. On the basis of these observations he regards catarrh of the middle ear with exudation as inflammatory and says that the treatment should be adapted to the inflammatory nature. By observation with the microscope for the tympanic membrane it is possible to differentiate from this form a catarrh of the middle ear without exudation, in which the changes in the eustachian tube are the same. The impairment of hearing in catarrh of the middle ear is not the result so much of negative pressure as of catarrhal changes. It is probable that changes in the internal ear likewise play a part in the impairment of hearing. Nevertheless, it is probable that occlusion of the eustachian tube and the resulting disturbance in the pressure may cause a temporary impairment of hearing. He thinks that increase in aviation, a vocation in which the patency of the eustachian tube is of great importance, may reveal more disturbances of this nature, and that more observations may permit a better differentiation between mere pressure disturbances and catarrhs of the middle ear.

EDITOR'S ABSTRACT.

PATHOGENESIS OF HEARING DISORDERS IN ASSOCIATION WITH CEREBELLAR TUMOR (CENTRAL BRAIN STEM DEAFNESS). H. BRUNNER, Monatschr. f. Ohrenh. **69**:1228 (Oct.) 1935.

Brunner points out that in previous discussions on mesencephalic deafness he showed that, although lesions in the mesencephalic portion of the secondary cochlear tracts readily elicit severe auditory disturbances, these tracts have considerable resistance to pressure which acts from the dorsal direction on the corpora quadrigemina. However, the author observed one case of large cerebellar tumor in which the brain stem had been considerably compressed. Since severe impairment of hearing resulted in this case, he decided to investigate the pathologic changes in the brain and the internal ear. On the basis of his microscopic studies he

reaches the conclusion that in some respects this case could be regarded as an instance of mesencephalic deafness, resulting from pressure of the tumor on the midbrain. Nevertheless, the clinical and anatomic aspects of the deafness of this patient differed from the typical manifestations of mesencephalic deafness. He mentions the following points: (1) absence of the typical symptoms of lesion of the corpora quadrigemina before the appearance of the deafness; (2) behavior of the labyrinthine function, for, whereas in mesencephalic deafness the irritability of the labyrinth is either normal or increased, in the case under consideration subnormal labyrinthine irritability was shown, and (3) severe impairment of the primary cochlear nuclei, which in cases of mesencephalic deafness were observed to be normal. Still, it must be conceded that the clinical manifestations indicate that pressure on the mesencephalic tegmentum was the chief cause of the auditory disturbance. Nevertheless, in view of the aforementioned points, the author designates the hearing disorder in this case not as mesencephalic deafness but as central brain stem deafness. He summarizes the anatomic and clinical aspects of central brain stem deafness.

EDITOR'S ABSTRACT.

Pharynx

FATAL PARENCHYMATOUS HEMORRHAGE FOLLOWING TONSILLECTOMY. E. URBANTSCHITSCH, Monatschr. f. Ohrenh. **69**:1187 (Oct.) 1935.

Urbantschitsch reports the clinical history and the results of necropsy in a case of fatal hemorrhage after tonsillectomy. Careful observation at necropsy did not disclose a vessel of even medium caliber that could be considered to be responsible for the hemorrhage. The hemorrhage took place in drops, unnoticed by the patient and by those who watched her. The use of a vascular ligature was therefore out of the question. Moreover, the patient never lost consciousness and only once, about five hours after the operation, stated that she felt as though the wound on the left side was bleeding. A real hemorrhage was never observed, in spite of repeated inspections. Thus, this case is a peculiar instance of fatal parenchymatous hemorrhage.

EDITOR'S ABSTRACT.

NEW TREATMENT OF TONSILLITIS. G. WOTZILKA, Monatschr. f. Ohrenh. **69**:1189 (Oct.) 1935.

Wotzilka directs attention to Waldapfel's studies in which that author demonstrated that substances which are injected into the lower nasal concha reappear in both tonsils. The connection between the concha and the tonsils is established by the veins that drain the cavernous tissue and communicate with the pharyngeal veins. To be sure, even when intravenous injection is made at other sites, small particles of the injected material appear in the tonsils but in much smaller quantities than when injection is made into the cavernous tissue of the nose. Waldapfel's observations on the connection between the nasal concha and the tonsils suggested to Wotzilka that it should be possible to introduce antibacterial medicaments into the tonsils by way of the lower nasal concha. As a medicament he chose a substance with which he had obtained good results in the treatment of otogenic meningitis. It contained methenamine and sodium salicylate in aqueous solution. After anesthetization of the lower concha he slowly injected 2.5 cc. of the substance. He obtained favorable results with this treatment in several cases of acute tonsillitis, in that the temperature fell rapidly and the pains in the throat and the difficulty in swallowing disappeared within several hours. Injection of the substance into the cubital vein was likewise followed by reduction in temperature, but it was less rapid and the local symptoms responded much slower than after injection into the nasal concha. In the conclusion, Wotzilka emphasizes that he wishes not to stress the value of the medicament he used but to direct attention to a new method of application of medicaments in the treatment of tonsillitis, namely, injection into the lower nasal concha.

EDITOR'S ABSTRACT.

TONSILLECTOMY IN THE ACUTE STAGE OF INFLAMMATION. A. JAUERNECK, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **26**:292, 1935.

Jauerneck points out that until recently it was generally considered advisable to postpone intervention on the tonsils until several weeks after recovery from tonsillar abscess or angina. Winkler, however, departed from this rule and advised tonsillectomy during the acute stage of tonsillar abscess but advised against this intervention in the course of angina without a suppurating process. The author calls attention to the fact that this differentiation made by Winkler has been corroborated by recent immunologic studies. Gangl-Battig reported in 1933 that the immunity, measured on the basis of values for phagocytes, is increased at the time the tonsillar abscess is at the height of its development and then decreases but that after another two or three weeks it increases again and reaches normal values in about five weeks. However, in angina without suppurative disintegration the phagocytic values are below normal at the height of the disease process, but after that they gradually increase and reach normal values in about five weeks. The author concedes that it is often difficult to determine whether there is a small abscess or progressive tissue infiltration without free pus. The phlegmonous processes which progress without the formation of pus are the most malignant, and early tonsillectomy is especially important in cases of this condition, for it is the only way to produce a wide opening of the phlegmonous area. In determining the advisability of tonsillectomy during the acute inflammatory stage, differentiation between phlegmon and abscess is unnecessary. The author advises tonsillectomy in the treatment of (1) deep and hidden abscess that cannot be located by incision and (2) abscesses that do not heal in spite of successful incision and apparently free discharge of the pus. In cases of the latter type there usually exist multiple abscesses, abscesses with several bays or phlegmonous processes. In accordance with these two indications, the author performed operation in ten cases, and the results were always good. As a third indication for tonsillectomy he considers the presence of incipient septic conditions during or after tonsillar abscess and angina in the course of which peritonsillar phlegmonous infiltration seems probable. Finally, he resorted to tonsillectomy in a number of patients in whom laryngeal edema threatened in the course of acute tonsillitis, with phlegmonous infiltrate under the tonsil. The phlegmon that develops during or after angina near the larynx and has a tendency to spread downward is always grave, and the author thinks that it is advisable to extirpate the involved tonsil. He found that after removal of the tonsil the laryngeal edema usually disappeared rapidly.

EDITOR'S ABSTRACT.

TONSILLECTOMY BY MEANS OF A SUCTION-SNARE INSTRUMENT. L. RIEGELE, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **26**:296, 1935.

Riegele describes an instrument that permits the removal of the palatine tonsils of an adult by means of a single instrument. The instrument represents the combination of a suction cup and a snare. By means of a suction bell the palatine tonsil is brought within the suction force of a water jet vacuum pump, and the tonsil, thus fixed, is then removed by means of a snare. The author gives a diagram of the instrument which clearly identifies the different parts, and he describes its operation. He shows that if the instrument is correctly manipulated the entire tonsil will be removed. Even if the tonsil is rather small or hollow and has a large plica triangularis, the latter is completely removed with the tonsil. Immediately after the tonsillectomy the tonsils should be carefully inspected in order to determine whether the surface of attachment to the tonsillar bed is intact, whether or not it has adhesions and whether the tonsil has been removed with the capsule. The intensity of the suction that is required to force the tonsil into the suction bell differs in the various cases. In cases in which there are only slight adhesions, a negative pressure of from 350 to 400 mm. of mercury is sufficient, whereas in cases in which adhesions are severe a negative pressure up to 60 mm. of mercury is required. If the suction is applied by means of a machine,

it is necessary to produce the negative pressure slower than if the jet of water is used. The author's experiences with his new suction-snare instrument cover a period of eighteen months. He states that his main purpose in devising this new instrument was to make tonsillectomy in adults as simple as that in children and young persons. In the course of performing tonsillectomy on fifty adults (aged between 18 and 68) he found that his suction-snare instrument permits clean tonsillectomy in cases in which the Ballenger-Sluder method fails. Postoperative hemorrhage is usually rapidly controlled. Especially noteworthy is the mildness of the after-pains in adults if the suction-snare instrument is used. In the concluding remarks the author stresses that his method differs essentially from Réthi's snare method as well as from the American suction method.

EDITOR'S ABSTRACT.

Nose

SUPPURATION OF THE MAXILLARY SINUS PRESENTING SYMPTOMS OF VASOMOTOR RHINITIS. K. M. MENZEL, Monatschr. f. Ohrenh. **69**:1169 (Oct.) 1935.

Menzel reports the histories of four patients with more or less complete obstruction of one side of the nose, profuse discharge of a watery secretion and frequent attacks of sneezing—that is, with the symptomatology of unilateral vasomotor rhinitis. In these cases, however, the disorder was caused by acute or subacute suppuration of the maxillary sinus. Nasal examination disclosed livid discoloration of the mucous membrane of the concha, which appeared to be inflated. Puncture of the maxillary sinus, however, resulted in the discharge of a considerable amount of mucous secretion in three cases. In the fourth case surgical opening of the maxillary sinus yielded caseous lumps. As the nasal symptoms began with acute suppuration of the maxillary sinus, they disappeared after the maxillary condition had been cured. As an explanation the author suggests that, in view of the rather nervous disposition of the patients, it may be assumed that the inflammation of the mucous membrane and the stasis of the secretion in the maxillary sinus caused irritation of the sensory terminations of the trigeminal nerve. The stasis of the secretion was produced by the complete closure of the involved side of the nose. The author concludes that in the four cases the condition, which became manifest with the aspects of vasomotor rhinitis, represents a special type of acute inflammation of the maxillary sinus.

EDITOR'S ABSTRACT.

CASE OF GOUNDOU-LIKE INVOLVEMENT, WITH POSTOPERATIVE ATTACKS OF VERTIGO. M. TAMARI, Monatschr. f. Ohrenh. **69**:1218 (Oct.) 1935.

Tamari concedes that the term goundou is usually applied to nasal tumors that occur in Negroes in the tropical regions. He emphasizes that if he applies the term goundou-like to the condition under consideration the histologic features are disregarded and only the external and roentgenographic aspects are taken into account, for, whereas in goundou appearing in Negroes in the tropics a productive inflammatory basis develops, the aspects of fibrous osteodystrophy predominated in the case reported. The previous history of the patient disclosed that during the war he had received a severe blow on the root of the nose with the butt of a gun. At that time he had been unconscious for two days, but no deformities of the face remained at the time. About ten years later the patient underwent bilateral excision of the lacrimal sacs for the relief of dacryocystitis. At that time thickening of the anterior wall of the right superior maxilla was observed. Two years after the removal of the tear sacs the patient observed slight bulging on each upper maxilla, which gradually increased but was not painful. The patient finally requested medical aid because of impairment in nasal breathing and loss of the sense of smell. Several months later he decided to submit to an operation on the right side of the nose, so that an air passage could

be made. In the course of the operation the bone was observed to be extremely hard and brittle. After tamponade and suture the patient suddenly had an attack of vertigo, with nausea and buzzing in the left ear. There were nystagmus of the second degree toward the right side and typical falling and past-pointing reactions toward the left. This attack, which resembled Ménière's disease, lasted one hour and recurred in somewhat milder form a number of times within the next three days. As far as breathing and the cosmetic effect were concerned, the patient was satisfied with the results of the operation, and six months later he requested that operation should be performed on the left side of the nose. After this second intervention the Ménière-like attacks recurred. The author states that regarding the origin of the Ménière-like attacks only conjectures are possible. Their concurrence with the operation suggests impairment of the labyrinth due to concussion, but the author thinks that the blows of the hammer alone do not offer a satisfactory explanation and that sudden circulatory disturbances or capillary hemorrhages in the labyrinth may have been responsible. EDITOR'S ABSTRACT.

SINUSITIS AND NEURALGIA: TWO FREQUENT SYMPTOMS DURING THE INFLUENZA EPIDEMIC OF 1935. EDITH HADASCHIK, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **26**:312, 1935.

Hadaschik observed a rather large number of cases of sinusitis and trigeminal neuralgia in the course of the influenza epidemic of 1935. The neuralgia was characterized by long duration and great intensity. It usually developed after sinusitis. The sinusitis was most frequent in men between the ages of 20 and 30, and it usually took a benign course and healed in response to conservative therapy. In three cases, however, the inflammation attacked the bone and necessitated operation. The author also discusses the histologic aspects of the sinusitis.

EDITOR'S ABSTRACT.

Miscellaneous

LIVING LACTIC ACID BACILLI IN THE THERAPY OF INFLAMMATORY DISEASES OF THE EAR AND NOSE. T. HÜNERMANN, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **26**:41, 1935.

Hünermann points out that the thought to employ living bacteria against pathogenic bacteria is not new but goes back to Metschnikoff and his followers. He also calls attention to the fact that lactic acid bacilli were used in otorhinologic practice twenty-five years ago. At that time American as well as French authors reported favorable results in ozena, sinusitis, rhinitis, otorrhea and rhinitis atrophicans. In recent years Griesmann again introduced the treatment with lactic acid bacilli. When a suitable preparation became available, Hünermann decided to try this treatment. The preparation used by him contains the various strains of Bacillus bulgaricus and Streptococcus lactis in such a symbiosis and under such bacteriologic conditions that autosterilization is impossible. The antagonism of the bacilli contained in the preparation to the pathogenic agents of the otic suppurations was first determined in experiments in vitro. Then the preparation was used in the treatment of eczemas of the ear, otitis externa, suppurative otitis media, pharyngeal catarrhs and suppurations of the maxillary sinus.

The method of treatment is as follows: If the preparation is to be applied in the ear, the ear is first cleansed with dry cotton, and then a gauze tampon that has been saturated with the preparation is introduced. After from five to ten minutes the tampon is removed and dry cotton is again introduced. This treatment is repeated daily. The patient can be told to drop 8 or 10 drops of the preparation into the ear, two or three times daily, let the fluid run out again after five or ten minutes and then close the ear with a tampon. To be sure, if the bone has

become involved, a radical intervention will be necessary. If the preparation of lactic acid bacilli is used, it is necessary to discontinue the use of disinfective substances.

Hünermann never observed undesirable effects from the use of the preparation. He observed that in postrhinitic pharyngitis the feeling of tension subsided, and in subacute suppurations of the middle ear he likewise observed favorable effects. However, in rhinitis atrophicans he was not able to influence the bacterial flora. After mentioning several other authors who employed the preparation with good results, he expresses the opinion that the efficacy of the preparation is probably due chiefly to the production of lactic acid, which in turn inhibits the growth and reduces the virulence of the pathogenic organisms. He admits that his material was too small to permit a definite evaluation, but he thinks his experiences justify further trials.

EDITOR'S ABSTRACT.

Society Transactions

AMERICAN OTOLOGICAL SOCIETY

Sixty-Ninth Annual Meeting, Detroit, May 28-29, 1936

FRANCIS R. PACKARD, M.D., *President, Presiding*

ABSTRACTED BY DR. ROBERT SONNENSCHEIN, CHICAGO

Thursday Morning Session, May 28

PRESIDENTIAL ADDRESS. DR. FRANCIS R. PACKARD, Philadelphia.

One of the most momentous meetings ever held by this society was that which occurred under the presidency of Dr. Thomas J. Harris, at Montreal, Canada, in 1926. At that meeting the final steps were taken for the incorporation of the society in order that it might be legally qualified to receive and expend funds for scientific research, and the committee on otosclerosis was able to announce that the Carnegie Foundation had granted to the society a large sum of money to carry on its work and that the movement was well under way to collect a large fund from other sources for the same purpose. At the same meeting Dr. Harris in a delightful presidential address gave a splendid summary of the history of otology in this country, especially of the work of the American Otological Society.

In the following year, 1927, Dr. Arthur B. Duel was president of the society. Without disparagement of the labors of any of those who were engaged in this great work, I think that I can say with justice that the society owes more to Dr. Duel than to any other person for the success of the movement to make a scientific study of that most baffling problem—otosclerosis. In his presidential address in 1927 Dr. Duel outlined the activities of the committee on otosclerosis, leaving it to Drs. Norval H. Pierce, Eugene A. Crockett, J. Gordon Wilson and James F. McKernon to report on the various details of the work which had been their charge.

Although a formal tribute will be paid to the memory of Dr. Duel in the transactions of the society, I cannot refrain at this point from speaking of the great loss which the organization has sustained by his death on April 11. Since his election to membership in 1904, Dr. Duel was always a most active and useful member of the American Otological Society. Devoted to its concerns, no man ever took a more alert interest in forwarding them. During the years that have passed since 1927 Dr. Duel not only continued his deep interest in the work of the Committee on otosclerosis but contributed greatly to scientific otology by his joint research with the late Sir Charles Ballance on the subject of facial palsy. Dr. Duel's charming personality added greatly to the joy of the meetings. In him otologists have lost a scientific leader and a dear friend.

Besides these two great leaders, Arthur Duel and Charles Ballance, the society has suffered the loss of many others during the past year—Dr. Campbell Smyth, Dr. J. J. Thompson, Dr. Franklin C. Capron, Dr. Edward B. Dench, one of the oldest and best loved members, and Dr. Albert Gray of London, England. In honor of these deceased fellows of the society I am going to ask the members to stand for a few moments in silent remembrance.

These two meetings of 1926 and 1927 mark an epoch in the history of the society. Thenceforth the tendency has been more and more toward giving up the meetings to papers and discussions on the scientific aspects of otology. Although there has been some adverse criticism of this policy, it seems to me that the advantages derived from such presentations are of incalculable benefit not only to those who attend our meetings but to the science of otology. There are few

who are engaged in clinical work who have either the necessary time or the ability to study the fundamental problems which underlie the science of otology. Not only have meetings in recent years afforded a medium by which those who are engaged in research in the physics, physiology and pathology of the organ of hearing can present and discuss their work, but I think that they have been of untold value to those who would otherwise be ignorant of the scientific foundations on which the otologist must depend if his practice is to be anything but empirical. In other words, otology is being placed in its rightful position as a science instead of an art.

With deep appreciation of the honor conferred on me when I was elected to serve as president at the sixty-ninth annual meeting of the society, I shall now proceed with the scientific business of the meeting.

SYMPOSIUM OF BONE CONDUCTION

REPORT OF THE COMMITTEE ON METHODS OF TESTING THE HEARING BY BONE CONDUCTION. STACY R. GUILD, PH.D., Baltimore.

(Dr. Stacy R. Guild, Baltimore, as chairman of the committee, presented the report by reading only the summary. With the consent of the society, reading of the complete report was omitted, since in mimeographed form it had been mailed to all members of the society in advance of the meeting.)

The general form of the committee's report has been determined by the fact that it is presented as a part of the symposium on hearing by bone conduction. Theories, new observations and the interpretation of data fall into the province of the contributions by individual investigators. The committee has attempted to present in a more or less logical sequence statements of the basic facts about conditions which affect the accuracy of clinical tests of hearing by bone conduction and the understanding of publications on the subject.

For the most part the facts presented in the report are not new, and many otologists know of the errors caused by failure to recognize these facts. Nevertheless, they neglect to observe the precautions that they know are indicated by the facts. The usual explanation offered is something like this: "I realize that my tests probably are not exact and that my findings may not mean much to others, but I have learned by experience how to interpret the results of tests I make with my own instruments under the conditions present when I use them. Therefore, I hate to change my methods."

While this attitude may be satisfactory for the individual otologist and his immediate work, there is no doubt that it is far from satisfactory from the point of view of the advancement of the knowledge of hearing by bone conduction. From the latter point of view, that of advancing knowledge, it is highly desirable that all otologists make their examinations by methods such that their reports when assembled can be utilized collectively in the study of the problems of impairment of hearing.

It is hoped that the research papers on the program today will arouse an active interest on the part of many otologists in the problems of hearing by bone conduction and that the work of these otologists will be aided by the outline of the status quo contained in this report. The committee believes that the methods suggested in the report for the avoidance of errors in testing hearing by bone conduction are practicable for adoption by otologists generally and that the observance of the precautions recommended should increase the value of clinical tests of hearing.

The statements of fact and the recommendations made in the report, which was mailed to all members of the society in advance of the meeting, are presented under six major headings, subdivided as follows:

I. Introduction

II. Suitable sources of sound for bone conduction tests
  (*a*) For threshold tests
  (*b*) For tests above threshold

III. The effect of room noise on the accuracy of tests of hearing

IV. The use of masking in clinical tests of hearing
  (*a*) Why use masking?
  (*b*) Which ear should be masked?
  (*c*) How can one ear be masked?
  (*d*) How can one produce satisfactory masking sounds?

V. Special points about the use of tuning forks
  (*a*) Technic of handling fork after it is struck
  (*b*) Activation of tuning forks; standard blows
  (*c*) Damaged and otherwise unsuitable forks
  (*d*) Where to place the stem of the fork
  (*e*) Calibration of tuning forks

VI. Special points about the use of audiometers with bone conduction receivers

Of the positive recommendations of the committee, the most important are: (1) that all tests of acuity of hearing be made in quiet surroundings; (2) that tests of hearing by bone conduction be made for as great a range of frequencies as is possible with the available instruments, always with due regard to the possible sources of error; (3) that suitable masking sounds be employed as a routine while making tests of the acuity of hearing by bone conduction. Practicable methods of producing satisfactory masking sounds are described.

ANATOMIC, PHYSIOLOGIC AND PATHOLOGIC OBSERVATIONS. STACY R. GUILD, PH.D., Baltimore.

(After a brief review by means of charts [lantern slides] of the anatomic structures primarily concerned with hearing and of the possible pathways by which sound waves may reach the organ of Corti during hearing by bone conduction, Dr. Guild presented evidence that one of the optional routes is of more importance than the others.)

Studies of sections of the temporal bone from patients whose hearing had been examined revealed a correlation between bone conduction time (the data with respect to bone conduction were obtained with a steel tuning fork of the frequency of 512 double vibrations per second) and the presence or absence of microfractures of the short osseous trabeculae that connect the medial end of the posterior wall of the external auditory canal to the inferolateral surface of the prominence of the horizontal semicircular canal. Microfractures of these trabeculae, with fibrous union of the fractures, are present in the sections from patients who had moderate or marked shortening of bone conduction time and normal or but slightly impaired hearing by air conduction of the sound; such fractures are not present or are incomplete in the sections from patients with a normal relationship between the hearing by air conduction and that by bone conduction or from patients with good hearing by bone conduction and impaired hearing by air conduction.

Hearing by bone conduction undoubtedly is impaired by lesions of the organ of Corti, the cochlear nerve or the auditory pathways of the central nervous system. But so far as revealed by the speaker's studies such lesions always impair the hearing by air conduction as much as that by bone conduction. The only lesions of the inner ear or of the middle ear that have been observed that are peculiar to ears with a disproportionate impairment of hearing by bone conduction are the microfractures of the osseous trabeculae already described.

A hypothetic explanation of the association of these facts is advanced. It is that sound waves entering the intralabyrinthine fluids from that part of the otic capsule to which these trabeculae are attached are more effective in stimulating the organ of Corti than are sound waves which are transmitted to the intralabyrinthine fluids through other parts of the otic capsule. Sound waves via the described trabeculae are transmitted to the perilymph from the posterolateral wall of the vestibule at a region adjacent to the footplate of the stapes and almost opposite the opening of the scala vestibuli into the vestibule. The facts warrant

the suggestion that this is the direction from which sound waves most effectively cause stimulation of the organ of Corti.

The suggestion is also warranted that poor hearing by bone conduction does not necessarily signify "nerve deafness;" that is, the lesion responsible for the impairment may be in the structures that transmit bone-conducted sound waves to the inner ear.

Experiments with animals designed to test the possibility that the bone conduction route for sound waves may be primarily via the so-called osseotympanic route were also reported. The Wever-Bray phenomenon was utilized to measure the relative intensity of the sound waves reaching the organ of Corti before and after interruption of the ossicular chain by operative removal of the lenticular process of the incus. Sound waves from the stems of tuning forks and from a hearing aid type of bone conduction receiver attached to an audiometer were transmitted to the heads of anesthetized cats through a wooden rod 12 inches (31 cm.) long, the end of which was held against the skin over the lateral end of the occipital ridge. The changes in electrical potential produced in the inner ear were picked up by an electrode in the region of the round window niche, suitably amplified and converted into sound waves by a telephone receiver. For the frequencies of 256 and 512 double vibrations per second the bone conduction thresholds were not impaired by removal of the lenticular process of the incus, but for the frequencies of 1024 and 2048 double vibrations per second they were slightly impaired on the average. As would be expected, this lesion regularly caused great impairment of the thresholds for air-conducted sounds of all frequencies. These experiments indicate that at least for the lower frequencies, the ones commonly used in clinical examination of hearing by bone conduction, the osseous pathway to the inner ear is of much more importance than is the osseotympanic route.

THE AVERAGE FOR AIR CONDUCTION AND BONE CONDUCTION IN THE BEST EARS OF PERSONS WHO CONSIDER THEIR HEARING NORMAL. DR. E. P. FOWLER and DR. E. P. FOWLER JR., New York.

In studying the audiograms of 1,064 hospital patients it was found that those who said they had no defect of hearing were often somewhat hard of hearing. Averages taken for the better ears of these persons showed good bone conduction for the lower tone ranges for all decades. In the later decades there was in addition a loss of hearing of high tones by bone conduction as well as by air conduction. Many of the older patients showed remarkably good hearing. Types of illness were studied, and the patients who had rheumatic fever were found to have the worst average normal hearing. The importance of testing the air and bone conduction, using the same units for each, was emphasized, as well as the fact that in all persons infections of the upper respiratory tract are important because they cause or increase lesions which cause deafness in the most used lower tone ranges not only in children but in older persons.

PATHOLOGIC CHANGES IN THE MIDDLE EAR IN PATIENTS WITH NORMAL HEARING AND IN PATIENTS WITH A CONDUCTIVE TYPE OF DEAFNESS. DR. LEROY M. POLVOGT and DR. JOHN E. BORDLEY, Baltimore.

The object of this study was to determine the common types and locations of the lesions in the temporal bone that are associated with a conductive type of deafness and a negative reaction to Rinne's test.

First, before attempting to analyze these lesions in the ears of patients whose hearing is better by bone conduction than by air conduction, it is necessary to become familiar with the pathologic changes found in the middle ear in patients with normal hearing.

The records of about 950 tested ears were studied; from this number only 63 met the requirements for the normal group, and 20 met the requirements for the group in which the time of bone conduction was better than that for air conduction.

The results of a comparative study of the histologic lesions in the middle and inner ears of patients with normal hearing and those with a conductive type of deafness and a negative Rinne reaction are as follows:

1. The most striking difference is the absence of any lesion of the ossicles in the group with normal hearing and the presence of one or more lesions involving the ossicular chain in every ear in the group with impaired hearing.

2. When the Rinne test gives negative results it is the result of interference with the normal movements of the ossicular chain by one or more lesions. This causes a reduction of the air conduction without changing the time of bone conduction.

3. The pathologic specimens on which these studies are based did not include the entire eustachian tube; so there is no information about tubal lesions in patients with normal or impaired hearing.

RESONANCE OF THE SKULL. DR. DOUGLAS MACFARLAN, Philadelphia.

It is obvious that by investigating hearing by bone conduction otologists seek to determine the true functioning of the end-organ of hearing unhampered by effects that might take place if the transmission apparatus of the middle ear has been impaired.

In testing bone conduction not only is sound conducted to the cochlea by direct transmission through the bone but a general resonance of the skull is set up, which may considerably alter the results. These effects are due to (1) lack of uniform density, size and shape of the halves of the skull and (2) filling of the sinuses, particularly the frontal sinuses and the antrums.

Lateralization of the sound from points along the midline (the Weber test) is greatly influenced by factors affecting resonance of the skull, and misleading conclusions may be drawn if resonance is not considered.

The contralateral reference of sound has not been satisfactorily explained. It does not occur with any great regularity or dependability. (Clinically, a fork placed on one parietal eminence should be heard in the opposite ear.)

The classic finding of reference of sound to the good ear in nerve deafness and to the bad ear in catarrhal deafness is not invariably reliable. In acute mastoiditis there frequently occurs the Weber-Schwabach paradox of MacKenzie, in which bone conduction is lengthened on the affected side yet reference from the midline is toward the good ear.

Lateralization from the midline may vary with the pitch used (alternating Weber, MacFarlan).

In bilateral otitis the value of lateralization is distinctly doubtful. Technics for testing resonance of the skull are described.

To minimize the absorption of the sound of the tuning fork by resonance when one is testing hearing by bone conduction the on and off method of applying the fork (Fowler) is urgently recommended. This technic allows contrasts of intensity of sound and reduces the tendency for the development of tone memory while testing.

(The paper was accompanied with charts and illustrations.)

BONE CONDUCTION, VIBRATION AND ELECTICAL STIMULATION. DR. HALLOWELL DAVIS, Boston.

The relation between bone conduction and the sense of vibration is considered. The high differential sensitivity for pitch, similar to that of normal hearing and far superior to the vibration sense, shows that bone conduction stimulates the true auditory mechanism.

Experiments involving direct electrical stimulation of the auditory mechanism are reviewed, and it appears that although direct stimulation of the auditory nerve is possible nevertheless a normal inner ear is essential for discrimination of pitch. This renders impossible the use of such electrical stimulation as an "artificial ear" in conditions involving loss of the organ of Corti. Bone conduction and electrical stimulation are useful diagnostic tools or aids to hearing only when used as alternative avenues of approach to the inner ear, which is essential for useful hearing.

CLINICAL OBSERVATIONS. DR. GEORGE E. SHAMBAUGH JR., Chicago.

Since the advent of the audiometer there has been renewed interest in tests of hearing and in evaluating the audiometer tests as compared with tuning fork tests, particularly with regard to bone conduction. The standard audiogram cannot be used to diagnose the type of deafness, since the shape of the curve is not characteristic. The standard curve supplemented by the bone conduction curve is not sufficient, because the standard curve has an element of bone conduction from the usual receivers, giving a false picture of the hearing for low tones in conduction deafness. Pure air conduction must be used, but this test is inaccurate and difficult to make. For practical purposes the tuning fork tests are quicker and easier to make and more accurate for diagnosing the type of deafness.

The prolongation of bone conduction noted in conduction deafness is due principally to the exclusion of outside sounds by the lesion, so that in a sound-proof room the reaction to the Schwabach test is normal in the presence of lesions of the conducting apparatus. The Schwabach test, therefore, should not be done in a sound-proof room. Moreover, the noisier the room the more clearly will a prolonged time of reaction to the Schwabach test be evident in early conduction deafness.

It has generally been assumed that different kinds of conduction deafness give the same reaction to tests of hearing provided the degree of deafness is equal. This is not true. If the cavity of the middle ear is filled with fluid, as in acute suppurative or catarrhal otitis media, there is impairment for the entire range of tones, the hearing of high tones being nearly as much impaired as that of the low tones. In lesions involving only the oval window, such as otosclerosis or traumatic perforation of the drum membrane, the loss of hearing involves predominantly the low tones, which are markedly depressed, with little or no lowering of the high tones. This difference in the reaction to tests for hearing in the two types of conduction deafness can best be explained by considering the mechanism of hearing by bone conduction.

While in most cases conduction deafness is easily diagnosed by the tests with tuning forks, one type of nerve deafness that involves chiefly the low tones may easily be confused with conduction deafness. This is the type associated with diplacusis, often with vertigo, in which the lesion is probably an inflammatory exudate in the labyrinth, an exudative labyrinthitis. The strongly positive Rinne reaction and shortened time of reaction to the Schwabach test serve to differentiate this type of nerve deafness for low tones from conduction deafness.

CLINICAL OBSERVATIONS ON BONE CONDUCTION. DR. W. J. MCNALLY, DR. T. C. ERICKSON, DR. R. SCOTT-MONCRIEFF and DR. D. L. REEVES, Montreal, Canada.

The object of this research was to investigate the acuity of hearing by bone conduction of a series of patients suffering from lesions of the inner ear or proved lesions of the cochlear nerve, such as tumor of the acoustic nerve or of some other intracranial nerve.

Much care was exercised in selecting the instruments for testing bone conduction. We were guided in our selection by the report of the Committee on Hearing Tests of the Royal Society of Medicine, London, for 1933.

A suitable routine of examination was adopted and was used in all cases except those in which the preliminary tests of hearing had been done before this investigation was undertaken. So far as possible a complete test of hearing was made before and after any operative procedure.

The complete examination consisted of: (1) taking of a careful history and inspection of the ears, nose and throat, (2) an audiogram (2-A Western Electric audiometer), (3) tests with the audiometer bone conduction apparatus of bone conduction and of absolute bone conduction for the notes of 256 double vibrations and 512 double vibrations, (4) tests with a tuning fork (512 double vibrations) of air conduction, (5) tests with a von Struychen monochord of air conduction and bone conduction and (6) a vestibular examination.

A standard length of vibration of the tuning fork was obtained by using a metal clamp, which was removed suddenly, after it had slightly compressed the prongs. The vibration time was recorded in seconds.

When there was a difference between the hearing in the two ears, the nontested ear was masked with a continuous current of air, an electric buzzer or a Bárány noise apparatus.

The patients examined fall into five groups for descriptive purposes:

Group 1. Thirteen patients were examined before and after encephalography and ventriculography to determine the effect of possible changes of intracranial pressure on the hearing. A change in hearing was not noted in any case.

Group 2. Fourteen patients had varying quantities of cerebral tissue removed from different areas, usually because of epilepsy or trauma. It was found that, as noted by previous observers, large portions of cerebral tissue may be removed from one side without causing any permanent damage to the hearing.

Group 3. There were 6 patients with cerebellar tumor. When the lesion was confined to the cerebellum, there was little change in hearing.

Group 4. In 9 patients with cerebral tumor no instance of loss of hearing was attributable to the lesion in the brain.

Group 5. There were 4 patients with perineural fibroblastoma of the eighth nerve and 1 with such a tumor of the fifth nerve. Usually there was almost complete deafness on the involved side, but it required masking to demonstrate the true condition. In a patient in whom the eighth nerve was not completely damaged, the hearing actually improved postoperatively on the diseased side.

Group 6. There are 5 patients with miscellaneous conditions in this group, including one in whom the eighth nerve had been sectioned for Ménière's syndrome and one in whom a loss of hearing for high tones was noted after removal of a tumor from the third ventricle.

Conclusions.—Of the three devices used to test bone conduction (the audiometer bone conduction attachment, the tuning fork and the monochord), one did not prove to be any more reliable than the others.

Whether bone conduction was tested on the audiometer for 512 double vibrations or for 256 double vibrations made little difference.

In 4 patients, each of whom apparently had one totally deaf ear, the bone conduction was greater in the poorer hearing ear.

The results obtained by the Weber test proved to be misleading.

The monochord served as a valuable check on the audiometer, but it is difficult to mask its effect from the nontested ear. The continuous current of air proved to be the most effective masking apparatus for the monochord.

These experiments suggest that the most accurate method of testing hearing at present is to combine the use of the audiometer, the tuning forks and the monochord for testing both air and bone conduction and that masking the nontested ear may be an essential part of the test.

During this investigation 52 patients were examined. In the complete report a short summary will be given of each case.

DISCUSSION

DR. ISIDORE FRIESNER, New York: The report of the committee on the testing of hearing by bone conduction contains a great deal of important information of such a type that it is difficult for an ordinary practicing otologist who is not a physicist to discuss it intelligently.

However, there are some suggestions in the report which I should like to review from the point of view of the practicing otologist. It seems to me that tests of hearing, which, of course, include tests by bone conduction, have a threefold purpose. First, and by far the most important, is diagnosis; secondly, adequate records, and, finally, for the future perhaps, a basis for the selection of that type of hearing aid most suitable to the patient under consideration.

It is particularly with regard to the first purpose of tests of hearing that I believe that otologists must still keep their feet on the ground, and therefore

I wondered at the statement that "we do not know how to interpret the information from certain bone conduction tests . . ." Otologists may not be able to explain these phenomena, but is not interpreting precisely what they have been doing for years with considerable success? Obviously, tests must be made under proper conditions and in a proper manner, but under such circumstances "shortened bone conduction should have a definite significance, a significance which has been of great value in diagnosis and which is quite as important as slight dulness at the apexes is to the internist.

I am glad, therefore, that the committee did not attempt the standardization of tests of bone conduction. I should be loath to abandon the tests which have been useful to me through the years, and I should be equally unwilling to discard the many records which I have accumulated, from which I have taught and which have been made on the basis of these tests.

R. L. Wegel, Ph.D., New York: There is a considerable amount of circumstantial evidence indicating that for any given pitch the same portion of the auditory nerve tissue is stimulated both by air and by bone conduction and probably in the same way. It does not follow that since the tines and shank of a tuning fork are vibrating at the same frequency, the pitch must be the same; yet this is substantially the case. Jones and Knudsen (1930) have shown that acuity of hearing by bone conduction bears a strong resemblance to that by air conduction in the way in which it varies with frequency. This is another of a number of indications that the vibration of all parts is substantially the same in both cases.

If the ear is tightly stopped by depressing the tragus with the finger, the sound from the air is reduced by about 35 decibels. A sound wave striking a body like the head is largely reflected, but that which is transmitted into it is in the neighborhood of 35 decibels below the level of that in the air. Pending a precise investigation of this problem, otologists may reasonably explore the consequences of the conclusion that on the basis of energy the ear is about as efficient in picking up sounds from the bone as from the air. In view of the great length of sound waves of acoustic frequency compared with the dimensions of the bony capsule, it does not appear useful to think of paths as definable channels through which sound energy flows to the end organ. It seems more reasonable to assume that stimulation takes place by a bodily vibration or distortion of the capsule or by modified air conduction. When resolved, this problem will probably be found much simpler than has been anticipated.

In regard to the testing of bone conduction, there is nothing that can be done with tuning forks which cannot also be done with properly made and properly used electrical vibratory apparatus. This new type of equipment will in fact be capable of much more flexibility, and on it must depend further substantial advances in the understanding of bone conduction. In the meantime, there is no reason for the otologist, in his practice, to abandon the tuning forks which he has been using successfully in diagnosis for years.

Dr. J. Gordon Wilson, Chicago: It is gratifying to see how successfully the younger members of the society have taken part in this symposium. I was pleased to hear Dr. Scott-Moncrieff say that at McGill University they have not dispensed with the monochord. At times when I am unable to decide whether a deficiency of hearing following a recent inflammatory lesion of the middle ear, especially of a patient beyond middle life, is due wholly to the conducting mechanism or whether the nerve is involved and to what degree, I find the monochord serviceable. The disadvantages of the monochord are well known, such as the difficulty of getting sounds of uniform intensity and the separation of the rub from the squeak.

The committee well deserves the thanks of the society for its report. The simplicity of its statements leaves little doubt regarding the significance of the various errors it uncovers and the means by which these can be rectified. The report contains a few general remarks which might well have been modified thus: "There is abundant evidence that few otologists make tests of bone conduction in conformity with any of the more precise methods that have been described in the

literature." Competent otologists are increasing in number, and so are those desirous of using more precise methods of testing. To blame otologists for not following authorities is a little beside the mark, for authorities differ, as is evidenced even in the present report. For instance, Politzer used a fork giving 256 double vibrations, not 512; Bezold used two forks, one of 106 and one of 460 double vibrations, his reason being that by this means he could differentiate combined lesions in the nerve and the middle ear. I use a fork giving 256 double vibrations and find it better than one giving 512, making sure that the patient differentiates the note from the vibration. Otologists fall down in the lack of standards to convey their findings to other examiners, in short, a common language.

The whole scheme for testing the acuity of hearing by means of a tuning fork depends on the identification of the time when the vibrations of the fork cease to be appreciated as sound. This is the identification of a subjective sensation, and subjective sensations are not always easy to follow to their minimum—not to mention after-sensations. So one has to depend on the help and intelligence of the patient as well as to guard against fatigue.

Having had some experience in trying to get a suitable sound-proof room, I believe that for the practicing otologist it is at present unattainable. He must be content with a quiet room. It is easy to get a moderately quiet room, but when one tries to tell a colleague how quiet the room is, one can assist by comparing the time in seconds between air and bone conduction in the patient with a normal reaction.

Dr. Robert Sonnenschein, Chicago: I wish to emphasize, as did Dr. Friesner and Dr. Wilson, the necessity of recognizing in a practical way the conditions under which otologists work. I am just as much in favor as any one of precision of tests and of accuracy in scientific work, but after all, otologists work in a noisy environment when doing the tests, whether that of Weber, Schwabach or Rinne. I pointed that out in the report of the modification that Dr. Minton and I made of the Rinne test about fifteen years ago. Even though from the physical standpoint some of these tests may not be perfect, from the practical standpoint they are valuable. It is known that when the tests are done in a certain way (and many of the conditions verified, as they were by Bezold, at autopsy) one can determine the presence of impaired hearing and the localization of the lesion—whether in the conduction or the perception apparatus or a combination of the two.

I am glad that the committee emphasizes the objective standard in the performance of the Schwabach test. In my paper read before the American Academy of Ophthalmology and Otolaryngology in 1921 and on other occasions I emphasized the fact that the method of doing the Schwabach test by placing the fork on the vortex or forehead and then comparing the patient's hearing with that of the examiner is fallacious, because one has to know definitely whether the examiner's hearing is normal, shortened or lengthened. By testing 100 or more normal persons at certain ages and determining how long on the average the fork used in a certain definite manner is heard one can simply compare that with the time the patient hears it.

Second, with reference to lateralization in the Weber test, of course otolaryngologists know (I emphasized that in 1911, in a long series of cases) that the Weber test is of value only when its results agree with and confirm those of other clinical tests. Of course, when there is a change in lateralization, for instance, when there is otitis media on the right side, and the patient lateralizes to that side, if the lateralization changes suddenly to the better-hearing ear and there are vertigo and other characteristic symptoms, the otologist knows that there is an extension to the inner ear—a labyrinthitis.

The report also states that the position and the forks used will cause variation in lateralization. I pointed that out in that same paper. Using the three forks, the a-1 (435 double vibrations), the weighted c-1, which is d sharp (154 double vibrations), and the A (108 double vibrations) fork, I found in from 11 to 15 per cent of cases that it does make a difference whether the fork is placed on the vertex or the forehead and some times which fork is employed.

With reference to Dr. Wilson's statement regarding the fork used by Politzer, when I worked in Vienna at various times years ago, the favorite fork was the so-called Vienna fork, which had the weight fixed definitely, with the pitch of d sharp.

Dr. Macfarlan spoke about resonance of the skull and cross-perception. In 1925 Schoen and Goldberger, of Vienna, published several papers in an attempt to determine how much resonance and cross-perception occurred and controlled the patient's hearing by means of the stethoscope tips in their own ears. The patient heard the sound by bone conduction and they heard it by air conduction. Bezold showed the matter by placing the stethoscope of Laënnec on a patient's head; when he on whose head the tuning fork was placed no longer heard it, the person whose ear was at the other end of the stethoscope still heard, proving that air conduction is longer than bone conduction in the normal ear.

Otologists know with reference to the cross-perception that when one places the tuning fork at any point not in the median line (for example, over the parietal region), as the speaker stated, the greater sound is heard on the opposite side, possibly owing to the union of vibrations. It has been shown that for some reason or other, when one places the tuning fork outside the median line, the skull divides itself into two portions, the smaller of which is that adjacent to the fork and the larger the opposite side; therefore, the resonance is greater on the opposite side and the sound is referred to that side.

With reference to sound-proof rooms, I agree with Dr. Pierce, Dr. Friesner, Dr. Shambaugh and others, for, as I have stated previously, otologists are testing patients who live in the same environment that they do. It is known that in a noisy room bone conduction is often apparently increased and that in most cases, as Politzer stated, the prognosis of a prolonged bone conduction is better than that of shortened conduction, because, except in association with otosclerosis, the prolongation of the bone conduction means impairment of the conduction apparatus, which is often not of great significance. However, in any case, even though in the sound-proof room there is no such thing as prolonged bone conduction, those who live in a noisy environment, if they have a relatively quiet room, can make the tests, and from those data, with the history of the patient, the examination of the ear, nose and throat and the correlation of the facts obtained can make a diagnosis. Then one knows the prognosis and knows which patients can be treated. But if one does use a relatively quiet room, I think one gets all the needed practical data. If one can work in a sound-proof room, certainly it is finer; the more accurate tests one makes the better.

Dr. F. R. Packard, Philadelphia: Before the discussion is closed, Dr. Harvey Fletcher, of New York, a member of the committee, wants to say a few words on something in the committee's report on the subject of masking.

Harvey Fletcher, Ph.D., New York: I want to say a few words about the report, and I wish to impose on the audience by bringing in an audiometer which has a masking device which may solve some of the problems wrestled with in the report.

With reference to the paper by Dr. Shambaugh, I confess that I was surprised, after hearing his paper and seeing how he used the audiometer, that he came to the conclusion that he could not use the audiometer but would have to use tuning forks. I mean by this that he determined the degree and kind of deafness of his patients by the audiometer and also whether their hearing had changed. I do not quite understand how he arrived at the conclusion he did.

I want to emphasize what Dr. Wegel said. It seems to me that the question of stimulation by a tuning fork or by any other device is a physical problem; it may be done any way provided that there is the same sort of vibration at the same point. One may use tuning forks if one uses them properly, and one must get the same results if one uses them as though using an audiometer. It is only a question of which is more convenient and which is more flexible. It is evident that those who have been using tuning forks and have built up a history and experience with them will want to continue with them. I think that is proper.

In my opinion the younger otologists who use the new audiometric methods will forget completely about the tuning forks as time goes on.

The committee discussed the testing with high and low tones, and I know that its significance has been discussed in this society before. There had been a tendency in the past to place most effort on testing with low-pitched tuning forks and high-pitched tones produced by the Galton whistle and other devices.

According to the present knowledge as to the mechanism of hearing, including that obtained from experimentation with animals that has been going on recently, it is evident that that is just the wrong way to do it. Those tones have the least significance of any, because in the high frequency region the variation from the normal is so large that when one gets a loss one does not know whether it is merely a variation from the normal or whether it means some lesion. If it were not for that fact, it might have more significance. With the low frequencies, according to the present conception, a 256 or 128 cycle tuning fork or tone will stimulate the basilar membrane right down to the end of it. If there is anything wrong with that or with any of the mechanism, it will show up for this frequency as well as for any lower frequencies. As one goes lower and lower in the scale of frequency, the difficulty of making the test increases. It was the opinion of the committee that it is not necessary to go below 128 double vibration; most of the members of the committee were inclined to say even 256. It is not necessary to test for frequencies higher than about 8,000, although that was not emphasized in the report, because every one did not agree to it. If I as a physicist were dictating, I should say that one would get most of the information about the mechanism by testing only between 200 and 8,000 cycles per second.

As to the masking device about which I spoke, it is attached to the new Western Electric audiometer, which I have here. An effort has been made to make this audiometer as simple as possible. Such an effort was also made in a design of the 2-A audiometer. But this one, I think, is made even more simple. There are only two dials, the pitch dial and the intensity dial. It works either on alternating or on direct lighting current and has no batteries. In order to save Dr. Fowler—who seems to be the only one who makes the important subtraction between bone conduction and air conduction—the task of making such subtractions, there is a dial here. This dial is slipped into place, and then one can read the deviation of bone conduction from the normal directly from the dial. This detachable dial has the advantage that as time goes on, if the American Standards Association which is studying this question changes the standards for normal hearing, all one has to do is to take the dial off and put on another one to meet the new requirement. This dial reads the same as that on the 2-A audiometer. With this dial on, one can read directly the deviation of the bone conduction from normal.

This is the masking device that gave me the excuse to present the audiometer, because it is on the subject. It makes it possible to produce masking without adding any equipment to the audiometer except a small switch, a coil and some simple resistance. The tone current from the audiometer comes into this circuit and divides into two parts. Part goes into the bone conduction receiver, which is held on the bone of the head for making the test, and the other receiver is held on the ear which it is desired to mask.

When the tone comes on, there is a continuous tone going in the masked ear, and the circuit is arranged so that the actual amount of stimulation given to it can never get through to the ear being tested, no matter where one turns the dials, because when the intensity of the sound is sufficient to be heard in the tested ear the sound is never sufficiently high in the masked ear to reach the tested ear. This circuit is arranged so that even though one goes through the entire range of frequency and intensity, it maintains the relative intensity, so that this is still true.

Some one will say: "How can one tell whether one is hearing in this ear or in that one, since the same frequency is used?" One could not tell by listening unless one of the tones was interrupted. When the testing tone is interrupted, if the interruptions can be heard, the listener knows that he is hearing this tone. This little button makes it possible to interrupt the testing tone without interrupting the masking tone. Thus, one determines the threshold in the usual way when masking is not used.

DR. ROBERT SONNENSCHEIN, Chicago: Is it permissible to ask Dr. Fletcher a question regarding that? How does he explain the curves shown by Dr. Sham-

baugh? (They were shown before the Chicago Laryngological Society.) How does he explain the fact that with the impairment of the conduction apparatus in one case and apparent impairment of the perception apparatus in the other he gets practically identical curves with reference to bone and air conduction?

HARVEY FLETCHER, PH.D., New York: I should prefer to refer that to an otologist like Dr. Fowler to answer. I should have to question whether they were the facts, if as Dr. Sonnenschein says, one is perception and the other conduction deafness, and the two conditions gave the same audiograms for bone and air conduction.

DR. ROBERT SONNENSCHEIN, Chicago: The tuning fork reactions, the whisper test and all other data one could possibly have were used.

HARVEY FLETCHER, PH.D., New York: I am not an otologist, but hundreds of tests have been made at the Bell Telephone Company Laboratories both on bone conduction and on air conduction. Dr. Fowler made thousands of them. From two complete audiograms, one on air conduction and one on bone conduction, I think the otologist can predict what he will get in every one of the other hearing tests.

DR. EMIL AMBERG, Detroit: One of the gentlemen spoke about the difference in the results obtained in a noisy room and in a quiet room. Another spoke about ideal conditions, and somebody spoke about repeated examinations. If repeated examinations are made, there must be doubt about the correctness of some of them. Then somebody said that the tests must be made under the same conditions.

I should like to put one little point into the discussion, which may perhaps not be of much value and yet may be of greater value than I imagine at present. That is the disposition of the examined person at the time of testing.

One of the essayists spoke about fatigue, which is an important factor. I want to call attention to Urbantschitsch's textbook (page 68). One can find illuminating facts in it. In the "Encyclopedia of Otology" there appears an article on fatigue by Max Goerke.

In experiments which are made in the psychophysical laboratories, great stress is laid on the fact that the experiments should always be made under the same conditions, even, I think, at the same time of the day, because as Politzer has said, hearing is worse at certain times. It is known that lack of sleep, hunger and various drugs affect the hearing. Experiments have been made on the vestibular apparatus with drugs. As far as I know, no systematic experiments have been made on the apparatus of perception. It is also important that neuroses be taken into consideration.

So the point I want to make is that in spite of the excellence and the painstaking accuracy of the report which was sent to us, it might not be amiss in the future to consult with a psychophysicist and use in the experiments the same precautions which are carried out in psychologic experiments.

DR. MAX A. GOLDSTEIN, St. Louis: There is one problem in this rather perplexing question of bone conduction that seems not to have been touched on by the various essayists. I refer to the relation of tactile impression to perception of sound.

Several years before Gault's introduction of the teletactor, I tried a practical experiment on congenitally totally deaf pupils at the Central Institute. These pupils had been trained to recognize variation in pitch with their finger tips and differentiation of pitch on the sounding board of the piano or organ and on the amplified loud speaker.

A boy of about 10 years was placed in the gymnasium on a high mattress so as to overcome the possibility of conduction of sound through the boards of the floor. He was stripped to the waist, and his hands were upraised, and through his skin, evidently, and the tactile sensation perceived by efferent nerves in the skin he could differentiate frequencies of sound—low, middle and high—either in individual notes or in chords struck on the piano or on the organ. What does that indicate?

Another practical experiment was made with children with congenital total deafness (they are good experimental material and fit in with this type of obser-

vation and investigation), who are good subjects for obtaining definite results in this problem of bone conduction. Congenitally deaf children who had been tested and who had been found to have an absolutely negative reaction to acoustic and static tests and so had no peripheral labyrinthine reaction were tested with tuning forks and with the audiometer on any part of the body, and they felt those differentiations of tone.

That brings to mind the classification of Dr. Guild in which in his diagrammatic representation he assumes this function, either by air conduction or by bone conduction, to be in the organ of Corti. I do not think that much is known about the organ of Corti, but otologists must not lose sight of that part of the cortex of the brain that is associated with the central perception of sound.

The congenitally deaf child who has a residuum of hearing is trained in this way. I check his hearing with the audiometer and find a small degree of sound impression in the low frequencies; after I have trained him for several years, I check his hearing again with the audiometer, and he demonstrates no more hearing, as far as the audiometric record is concerned, than he had when he started.

Lorente de Nó, at the Central Institute for the Deaf, in the last few months came to the conclusion, after making laboratory tests of afferent sound impulses in the comparison between a sound traveling over the sciatic nerve and one traveling over the auditory nerve, that the time required for that sound perception centrally is exactly the same. In other words, the auditory nerve, like the sciatic nerve, is simply an electric conducting wire.

Dr. Horace Newhart, Minneapolis: On this auspicious occasion, when so many internationally known acoustic engineers are present, I wish to ask two questions of practical interest to the practicing otologist.

The first is regarding the sound-proof room. All those present know that no otologist or practically none aspires to the possession of an absolutely sound-proof room. Many otologists, however, are making the attempt to secure an acoustically treated room in which the noise level is so reduced as to make reasonable accuracy possible by eliminating the most distracting, masking noise.

In this report I note the suggestion that it is better to line such rooms with a hard surface than with sound-absorbing material. It seems to me that this at once introduces elements of error in making the ordinary tests, in that an increased amount of noise is produced by the reverberation of the noises that penetrate within that acoustically insulated space.

The second question is regarding the interposition of a thick cushion of sponge rubber between the auricle and the air conduction receiver in testing acuity for air conduction. This necessarily makes a considerable difference (according to the degree of pressure which the patient makes on the receiver) in the distance between the vibrating diaphragm of the telephone receiver and the tympanic membrane. It seems to me that an appreciable element of error is thus introduced. Does the advantage from this interposition of a thick layer of sponge rubber to eliminate bone conduction compensate for the possible element of error thus produced?

Harvey Fletcher, Ph.D., New York: Dr. Newhart asked a question. The committee made a report on the question of sound-proof rooms and made a recommendation, as he pointed out, that the inside of the room be constructed with hard walls. I do not think the committee meant to make it uncomfortably hard; it wanted to point out that it is not necessary to have it completely filled with absorbing material as has been done in the past.

However, I think that there is some advantage in having absorbing material on the inside, because it helps to reduce the noise. There is a disadvantage in that it puts the patient in a little different environment, and that is the point the committee wished to bring out.

I desire to say a word about the question of testing in a noisy room and making correction for it. The corrections cannot rightly be made. To illustrate what I mean consider the following. If I were in a noisy room with a person who is hard of hearing I might be considered the patient instead of the hard of

hearing person, because I would be more handicapped than he. In some tests he could hear better than I. That is an exaggerated case, but the same principle holds for all degrees of noise. If any noise is present, one cannot differentiate between small amounts of deafness and normal hearing. The more noise there is, the larger the range of deafness which cannot be detected.

STACY R. GUILD, PH.D., Baltimore: May I answer Dr. Newhart's two questions? Most sponge rubber ear pieces are not satisfactory for the reason he gives. I have used at Baltimore a kind originally selected by Dr. Bunch. Within the last two years I have discovered another satisfactory one. I check the different ones by actually testing normal persons without and with the ear pieces and find that the use of certain sponge rubber ear pieces on the receiver of the audiometer does not cut down the threshold readings obtained. Selection must be on the basis of actual trial with persons with normal hearing until one is found that has the same acoustic impedance as does the receiver applied directly to the ear. Sponge rubber ear pieces do help to exclude extraneous noises.

With respect to the hard walls, I call attention to the wording of the report: "The committee recommends also that the walls of this room or booth be of hard material, because many patients are so disturbed by the absence of reverberation in a padded room that they do not cooperate well in the tests."

Any one who has ever been in a completely sound-proof padded room will appreciate the feeling of a patient who is not accustomed to it.

I wish to question two features of this instrument for masking. First, I do not believe that the use of the same frequency is as good for masking purposes as is a mixture of tones. One reason for this belief is familiar to all otologists who have used the two fork test.

However, since this comes from the Bell Telephone Laboratories and has received the support of Drs. Wegel, Fletcher and others of their group, who know more about the effect of pure tone masking than I do, I hesitate to speak further about it at this time. I do feel, however, that considerable experimenting should be done with that method of masking for clinical purposes before it can be recommended.

The second thing about the instrument shown by Dr. Fletcher is that it maintains a constant differential between the intensity of the masking sound and that of the test tone. That is all right for certain types of impairment of hearing, but for patients with a unilateral conductive lesion, unilateral otosclerosis or chronic otitis one needs a different intensity for masking than when both middle ears are normal. The differential in intensity at which sounds "cross over" from one ear to the other is much affected by such conditions; therefore, a masking apparatus should provide means of changing the intensity of the masking sound with respect to the test sound.

HARVEY FLETCHER, PH.D., New York: This is only for bone conduction masking.

STACY R. GUILD, Ph.D., Baltimore: All masking for clinical purposes must be by air conduction. One cannot use bone conduction for masking, because both ears are stimulated. That is in the report of the committee, and I am thoroughly convinced that it is so.

I was much interested in Mr. Wegel's comment regarding a 35 decibel loss when a plane wave front strikes the integument. I did not call attention (it will show in the published article) in my first lantern slide to the arrow from the air side of the chart direct to "integument" and then up along the bone conduction side of the chart. Elaboration of that diagram, with consideration of the coefficients of absorption and so forth for soft tissues, should be done by some one with Mr. Wegel's special qualifications. I feel that there is an important tie-up between bone conduction and air conduction and that this relationship may explain many things.

DR. DOUGLAS MACFARLAN, Philadelphia: I think a good deal would be missed at this meeting if one did not remember Dr. Guild's paper and his description of the discrepancy between air conduction and bone conduction in the impairment of hearing associated with fractures. I think they will lead far if they are followed.

As to Dr. Fowler's paper, I think that there is need of amplifying the clinical data on hearing by bone conduction by the report of the physical analysis of these receivers. I think that otologists often do not know enough about the physical characteristics of the instruments they are using, and there are few data on the physical condition of the bone conduction receivers now in use. Of course, many otologists are using different types.

Dr. George E. Shambaugh Jr., Chicago: Dr. Fletcher criticized me for using the audiometer to compare improvement of hearing when I said it was not worth anything. I did not say the audiometer is not worth anything for comparing the improvement in hearing. It is more valuable than any other test available for that. For diagnosis, I do not believe that it is as valuable as the tuning fork test.

For example, the last patient presented had diplacusis and nerve deafness for low tones. I maintained that with the audiometer curves, no matter how they are made, one could not by comparing bone conduction with air conduction diagnose this condition as nerve deafness for low tones. There is a false impression of hearing for the low tones in the standard curve. The patient could not hear the low tones at all by air conduction, as shown by the tuning fork test.

Harvey Fletcher, Ph.D., New York: Was the curve for bone conduction plotted from the dial, or were subtractions made?

Dr. G. E. Shambaugh Jr., Chicago: It was plotted without subtractions.

Harvey Fletcher, Ph.D., New York: Would the subtraction show what you want?

Dr. G. E. Shambaugh Jr., Chicago: No. It would show a negative Rinne reaction. Yet the fork showed that the Rinne reaction was positive. I maintain that the audiometer could not have shown this to be nerve deafness for low tones. In a case of ordinary deafness it is easy enough, with the audiometric curves, by comparing air and bone conduction to make the diagnosis of nerve deafness or conduction deafness. It is in these borderline cases that diagnosis is most important and most difficult. Therefore, I think tuning forks are necessary.

(Slide) Here is a patient with a unilateral dead ear. I know that he has a dead ear, for I removed the labyrinth because of suppurative labyrinthitis. The bone conduction curve for the low tones shows that they are heard through to the other side. The tone from the standard audiogram receiver, I believe, is also heard around through the head to the other side by bone conduction. I believe that it is heard through the head by bone conduction from the standard receiver, which fits close to the head.

C. C. Bunch, Ph.D., St. Louis: My idea in using masking was to prevent hearing in the good ear in cases of complete deafness of one ear. If one starts at the right ear, it shows the hearing in the left ear, because one has not masked out the hearing in the left ear.

Dr. George E. Shambaugh Jr., Chicago: That is correct; masking is necessary. Even with masking I think one gets false hearing in the bad ear in case of conduction deafness, because I believe that the patient is getting some element of bone conduction from the standard receiver, and one is not really measuring the air conduction.

C. C. Bunch, Ph.D., St. Louis: Evidently in this chart Dr. Shambaugh is not using sufficient masking.

Dr. George E. Shambaugh Jr., Chicago: I did not use any masking.

Dr. R. Scott-Moncrieff, Montreal, Canada: As regards the matter of absorption of sound by soft tissues, it may be of interest to say that in one of our cases, an instance of cholesteatoma with epilepsy, the patient had had a radical operation on the mastoid. A day or two after the operation was done, the bone conduction was tested, with the fork placed on the bare bone in one instance and on the edematous tissue just back of the open wound in the other. There was about three seconds difference between the two, which we thought was rather striking. We felt that there should be much more difference due to the edema.

Thursday Afternoon Session, May 28

REFERRED PAIN IN THE EAR. DR. PERRY G. GOLDSMITH, Toronto, Canada.

Dr. Goldsmith stated that the subject was being discussed for the second time since the society's organization.

The anatomic connections and reflex pathways that may be associated with referred aural pain were reviewed. An attempt was then made to review systematically the possible lesions that might cause reflex stimuli from the various regions. A number of patients, with short case histories, were presented to illustrate the various points that had been discussed.

DISCUSSION

DR. GEORGE M. COATES, Philadelphia: I shall not attempt to discuss the almost innumerable causes of referred pain in the ear but shall simply call attention to one particular cause, which Dr. Goldsmith mentioned, I think, but did not stress.

At the meeting of the American Medical Association in Kansas City a couple of weeks ago, an excellent paper was read by Dr. James Costen, of St. Louis, on the effect on the ear of closing of the bite. Dr. Costen showed that frequently when there is malocclusion due to dental deformity, particularly to loss of teeth on one side, the temporomandibular joint becomes subluxated or at times eroded, and these erosions and malformations can be shown well by the roentgenogram. I have had some experience with this condition.

These erosions, particularly those in the joint from closing the bite, do cause referred pain in the ear. They also at times cause a conductive type of deafness and particularly tinnitus aurium.

Dr. Costen's idea is to open the bite, which can be accomplished in a diagnostic way by putting a couple of small cork disks between the upper and the lower jaw on the affected side to see whether temporary opening of the bite will relieve the symptoms. If there are any signs of improvement in the patient's condition, it is the task of the dentist to put on "skids" or to build up the teeth on that side, so as to equalize the pressure on the two sides.

DR. J. GORDON WILSON, Chicago: If no abnormal condition can be found by aural examination, information as to the source of the referred pain can be obtained from observation of the painful area, for it is obvious from what Dr. Goldsmith has said that the location of this area will vary with the distribution of the nerve.

One point perhaps not in direct relation to Dr. Goldsmith's paper but of importance is the cause of the pain that may arise from a loud noise. Although it is assumed by some that it has a direct relationship to the audiometric nerve, there is no evidence to support this assumption. Certainly a part is related to painful sensations arising in the membrana tympani, which is fundamentally a protective structure, As I pointed out several years ago, and a part may be due to excessive contraction of the tensor tympani, as Hallpike recently suggested.

DR. ROBERT SONNENSCHEIN, Chicago: Probably Dr. Goldsmith mentioned in his paper, but did not have an opportunity to read, that in tuberculosis of the larynx an ulcer on the posterior wall will often cause pain that is referred only to the ear and not to the throat.

DR. HARRIS P. VAIL, Cincinnati: There are a few things in Dr. Goldsmith's paper that I might criticize, if I may use that word. One is his failure to give more credit to the great superficial petrosal nerve for the transmission of sensory impulses. I think that it has been pretty well shown by anatomists—Koontz, Fenton and others—that the great superficial petrosal nerve is a sensory nerve in addition to any sympathetic function which it may perform. I think this can be shown by studies on tic douloureux after section of the sensory roots. All sensation in the head is not abolished. These people have deep sensation.

I have at present a patient who has complete anesthesia over the face as a result of section of the posterior root for trigeminal neuralgia, yet she has to have her posterior sinuses cocainized when the sphenoid is washed out. I know that cocaine applied to the sphenoid sinus will relieve some types of headache, but whether they arise from the great superficial petrosal nerve is a little hard to say.

The microscopic picture of the great superficial petrosal nerve seems to show that it is a sensory nerve. I am willing to grant that the sphenopalatine ganglion is entirely sympathetic.

Dr. Perry G. Goldsmith, Toronto, Canada: About twenty-six years ago Dr. Wilson published an interesting article, which he was good enough to send me. Any statement he makes on the innervation of the drum membrane may be accepted without question.

In tuberculosis of the larynx or any malignant condition it is not uncommon in the advanced stages to have pain in the ear, and that pain is usually a bad symptom.

I am not going to enter into the question of the sphenopalatine ganglion. I think it is an enormously overrated thing, and I hope that the subject will be buried.

Some Observations of Facial Palsy. Dr. James A. Babbitt, Philadelphia.

Major progress in overcoming palsy of the seventh cranial nerve has occurred in the past forty years. There seem to be four principal phases, characterized by the following procedures: (1) research on the nature and physiology of regeneration of the nerve, (2) anastomosis of the distal segment with other motor trunks, (3) transplants of temporal muscle and fascia to relieve deformity and (4) decompression and substitution of transplanted nerve tissue in the fallopian canal. Two cases of facial palsy of about twenty-five years' standing are discussed, with the results in one following transplantation of temporal muscle in a postauricular defect and, in the other, transplantation of temporal muscle and fascia to the region of the eye and the mouth, supplemented by a transplant from the fascia lata (Dr. Ivy). These results are illustrated by moving pictures. Conclusions are aided by the findings of Ballance, Duel, Greenman and Fogliotti.

DISCUSSION

Dr. John R. Page, New York: Dr. Babbitt states in his paper that according to Cushing the first nerve anastomosis was performed by Faure in 1898. I am sure that he will find in the article by Sir Charles Ballance and Arthur Duel published in the Archives of Otolaryngology for January 1932 that Sir Charles Ballance united the facial nerve end to end with the spinal accessory nerve in 1895.

In his first case the essayist says the improvement in the facial muscles must have been due to the large pedicled graft of temporal muscle that was used to fill the mastoid cavity and which, fortunately for the facial muscles, extended 2 cm. below the lobe of the ear. It is almost fantastic to believe that innervation of the muscles of the face came from the lump of temporal muscle that was used to fill the mastoid cavity, but such must certainly have been the case. In Rucker's article entitled "Surgical Treatment of Facial Paralysis," printed in German in 1933, he stated that the experiments of Erlacher show that the motor nerve fibers of implanted muscle grow into the injured muscle and form a new innervation and that on the basis of these findings Rosenthal implanted broad flaps of the temporal muscle and the masseter muscle into the muscles of the eye, cheek and angle of the mouth. He furthermore stated that muscular neurotization presents distinct advantages as compared with neuroplastic procedures. In Dr. Babbitt's first case, however, no strips of muscle lay in contact with any of the facial muscles themselves but the end of a lump of muscle supplied by the fifth nerve extended from the mastoid cavity and lay somewhere near the degenerated branches of the seventh nerve in the region of the parotid gland. In the second case the idea of taking strips of fascia lata and making swings of them to support the sagging muscles of the affected side is particularly useful in conjunction with grafts of muscle and nerve in cases of long-standing paralysis to oppose the pull of the active muscles on the opposite side and to give better opportunity for the graft to take effect.

According to the abstract of Rucker's article referred to, among the older attempts at myoplastic work were those of Gomoiu, who used the sternocleido-

mastoid, and Jiam, who employed the masseter. Lexer likewise used the masseter for a flap split into two tongues, one being brought above and one below the angle of the mouth and attached through a small incision at the angle, with the corner of the mouth drawn up in an overcorrection that he deemed necessary. In cases of paralysis of the upper branches of the facial nerve he used also a flap from the temporal muscle and drew it through the undermined skin to be anchored by sutures under the paralyzed eyelid.

An interesting and encouraging statement in the literature is the reference to the experiments of Erlacher, which are said to show that the motor nerve fibers of implanted muscles grow into the paralyzed muscle and form a new innervation of it. Today there has been shown an example of an innervation of paralyzed muscles not from a strip of live muscle in contact with them but from muscle that was barely in contact with the trunk of the degenerated nerve that supplied them.

DR. JOSEPH C. BECK, Chicago: The two cases reported here are encouraging, particularly the first one. It would have been interesting to have the neurologic diagnosis, that is, the reaction of degeneration. In cases of this type, Faure, the first man who operated for facial paralysis, so far as I know, although I do not wish to dispute the later investigation, said that such an association is not possible after a reaction of degeneration, that is to say, after a true fibrosis of the paralyzed muscle is present; therefore, I should like to have that point cleared up by Dr. Babbitt.

During my years in practice I have operated on 17 patients with facial paralysis by anastomosis, first by anastomosis with the spinal accessory nerve, as first described by the late Dr. John B. Murphy, of Chicago. That method was discarded because of the movement of the face associated with the movement of the arm. The anastomosis with the facial and the hypoglossal nerve brought end to end and end to side gives a much better result. A good many years ago I reported in the *Annals of Otology, Rhinology and Laryngology* a case in which the patient was successfully treated by end to side anastomosis of the facial and hypoglossal nerves, so that the tongue was not affected.

The operation, which apparently is not being discussed here today, namely, that of the late Dr. Duel and Sir Charles Ballance, I think should at least be mentioned in an encouraging manner.

Regarding the work of transplanting fascia and muscle, I have had considerable experience. The external masseter muscle split, with the free end brought into contact with the angle of the mouth, gives the best result of any that I have tried. The temporal muscle when employed is thinned out, and it is difficult to perform the operation on that account.

DR. EDMUND P. FOWLER JR., New York: The movies of Dr. Babbitt's first patient show typical results of repair of the seventh nerve. I should like to ask whether the movement in the face of this boy was produced in an effort to move his face, as for a smile, or by biting his teeth together. If it was not produced by biting his teeth together (that is a movement which is activated by the fifth cranial nerve) I should suggest that the return of his facial movement was due to releasing the neurons of the seventh nerve in the mastoid wound. That is, it is the revision of the mastoid and the removal of the neuroma in it which are responsible for this boy's return of facial function and not the transplant of temporal muscle.

DR. JAMES A. BABBITT, Philadelphia: Any recognition of priority due Sir Charles Ballance in the nerve anastomosis operation should be adjusted. There seems to have been some confusion in the literature as to chronology.

In answer to Dr. Beck, I would say that the neurologic reports were definite. I am not entirely clear as to Dr. Fowler's suggested explanation and cannot state definitely as to the patient's mouth closure on producing facial movements. He made these as active as possible in answer to a request.

(To be concluded)

Directory of Otolaryngologic Societies *

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Collegium Oto-Rhino-Laryngologicum Amicitiæ Sacrum

President: Prof. Gunnar Holmgren, Karolinska Mediko-Kirurgiska Institutet, Stockholm, Sweden.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg, 143 Groningen, The Netherlands.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Robert F. Ridpath, 1737 Chestnut St., Philadelphia.
Secretary: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Place: Atlantic City, N. J. Time: June 7-11, 1937.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Wells P. Eagleton, 15 Lombardy St., Newark, N. J.
President-Elect: Dr. Frank E. Burch, 408 Peter St., St. Paul.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

American Bronchoscopic Society

President: Dr. Joseph C. Beck, 185 N. Wabash Ave., Chicago.
Secretary: Dr. Lyman Richards, 319 Longwood Ave., Boston.

American Laryngological Association

President: Dr. W. B. Chamberlain, 10515 Carnegie Ave., Cleveland.
Secretary: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Atlantic City, N. J. Time: May 1937.

SECTIONS:

Eastern.—Chairman: Dr. D. C. Jarvis, Quarry Bank Bldg., Barre, Vt.
Southern.—Chairman: Dr. Charles D. Blassingame, P. & S. Bldg., Memphis, Tenn.
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Mid-Western: Dr. Frederick A. Figi, 1013 First St., S.W., Rochester, Minn.
Western.—Chairman: Dr. David R. Higbee, 3245 Fourth St., San Diego, Calif.

American Otological Society

President: Dr. Edmund Prince Fowler, 140 E. 54th St., New York.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.